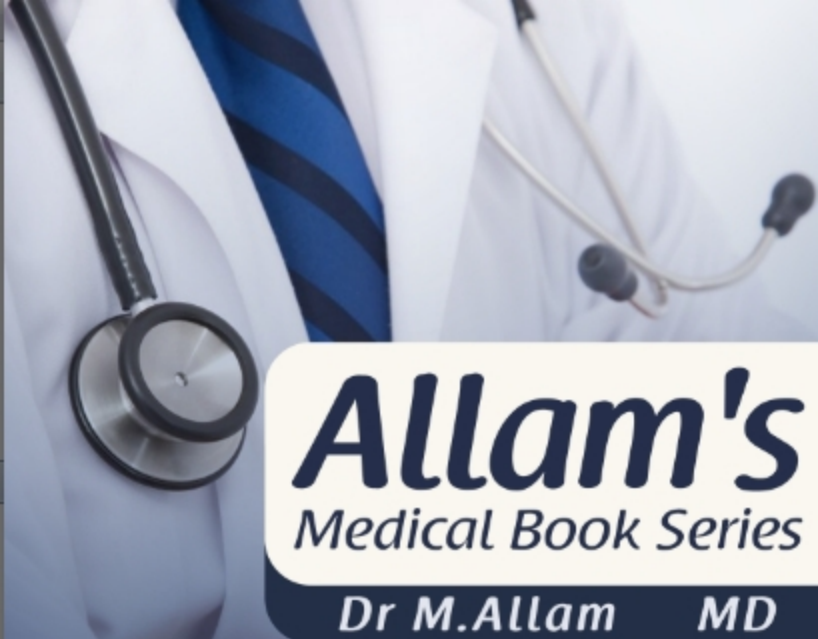




Allam's

Medical Book Series

Dr M.Allam MD

Part 1



General
history & examination



Cardio Vascular
System

VISIT...

LANZAROTE
Caliente.COM

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HISTORY

GOOD HISTORY = GOOD DOCTOR

1. Personal History
2. Complaint
3. History of present illness
4. Past history
5. Family history
6. Socioeconomically state
7. In females (Menstrual and obstetric history)



(1) Personal history

- | | | | | | | |
|---------|--------|--------|---------------|------------------|--------------|----------|
| 1. Name | 2. Age | 3. Sex | 4. Occupation | 5. Marital state | 6. Residence | 7. Habit |
|---------|--------|--------|---------------|------------------|--------------|----------|

1. Name اسم ثلاثي

- To be familiar with the patient.
- Sex identification.
- False errors may occur when two patients with the same name have been under treatment in the hospital simultaneously.
- For follow up.

2. Age

Diseases common in children

- Rheumatic fever.
- T.B.
- Hemolytic anemia.
- Acute leukemia.
- Poliomyelitis.
- Duchenne myopathy.
- Friedrich's ataxia.

Diseases common in old age

- Carcinoma.
- Atherosclerosis and coronary artery disease.
- Cor pulmonale.
- Chronic lymphatic leukemia.
- Multiple myeloma.

N.B.

Tumors occur in children:

- Wilm's tumor of the kidney.
- Retinoblastoma.
- Medulloblastoma.

3. Sex

Diseases common in male	Diseases in female
- I.H.D. – Hemophilia, Duchenne (X-linked) - Bronchogenic carcinoma	- S.L.E – Thyrotoxicosis - Myxoedema. - Chorea – Myasthenia.

4. Occupation

- lead worker:** anemia – nephropathy – peripheral neuropathy.
- Glass workers (Silicosis):** interstitial pulmonary fibrosis (IPF).
- Asbestosis:** IPF – bronchogenic carcinoma or mesothelioma.
- Farmers:** bilharziasis – farmer's lung.
- Medical staff:** infection – X-ray irradiation (bone marrow depression – sterility).

5. Marital state

Ask about

- Duration of marriage.
- Number of children.
- The age of the youngest one.

6. Residence & Address

May reflect socioeconomic condition and may occasionally point to a certain disease e.g.

- Country:** Bilharziasis – exposure to animals (Brucellosis) – insecticides.
- Towns:** anxiety – hypertension – IHD.
- Sharkia:** Filariasis!!!

7. Habit

Special habit is a habit that makes the patient more susceptible than others to a certain disease.

a) Smoking

Ask about number of cigarettes/day and duration + type of smoking (pipe, cigarette)

Smoking index = No. Of Cigs/day X duration in years

- Mild → < 100.
- Moderate → 100 – 400.
- Heavy → > 400.

Calculating pack years of smoking

20 cigarettes = 1 packet

$$\frac{\text{No. of cigarettes smoked per day} \times \text{No. of years smoking}}{20}$$

For example,

A smoker of 10 cigarettes a day who has smoked for 15 years would have smoked:

$$\frac{10 \times 15}{20} = 7.5 \text{ pack years}$$

Hazards of smoking

(1) Chest	(2) C.V.s	(3) G.I.T.	(4) Other complications
- Chronic bronchitis, emphysema (COPD). - Bronchial asthma. - Cancer lip and tongue. - Post operative pneumonia.	- Arrhythmia. - Hypertension. - I.H.D. - Peripheral vascular disease.	- Peptic ulcer. - Cancer esophagus - Cancer stomach.	- Cerebrovascular disease. - Cancer bladder. - Intra uterine growth retardation. - Tobacco amblyopia.

b) Alcohol

Ask about the amount / day.

10 gm alcohol = **30** ml of whisky = **100** ml wine = **250** ml beer.

Excessive alcohol intake

Male consumes > 21 unit / week & female consumes > 14 unit /week.

Hazards of alcohol intake

(1) G.I.T.	(2) C.N.S.	(3) C.V.S.
- Mallory-weiss syndrome - Alcoholic fatty liver - Alcoholic hepatitis then cirrhosis. - Acute hemorrhagic pancreatitis.	- Wernicke's - Korsakoff syndrome (amnesia + confabulation). - Polyneuropathy. - Proximal myopathy. - Optic atrophy. - Hallucination - delirium and coma.	cardiomyopathy

N.B.

- Familial tremors are important in small amount of alcohol.

(2) Complaint**C/O + DURATION**

إيه اللي جابك المستشفى؟؟

- By the patient own words – No medical terms.
- As short as possible (one complaint is enough).

هنشوف مثال بسيط على كده

نقول مثلاً the patient is complaining of shortening of breathing of **1 week** duration

في المثال ذكرنا إن المريض يشتكي من كَرْشَة نفس ، وده من كلام المريض

ومستخدمناش مصطلحات طبية ، وكمان كلام مختصر زي ما أنت شايف

وكمان كتبنا في الآخر ال duration ، اللي كانت one week

(3) HPI

1. Analysis of complaint.
2. Symptoms of the related system.
3. Other systems.
4. Investigation & treatment (related diseases).

Characterized by:

- As long as possible
- Contains medical terms
- In chronological arrangement
- In the form of a story.

For analysis of any symptom

- Onset / Course / Duration.
- Association.
- What ↑ and what ↓.
- Effect of TTT.
- Date of last attack.

N.B.

- For analysis of pain = 8 as previous +3 (site, radiation and character)

(4) Past history

Disease	Operative	Drugs
• D.M. • Hypertension. • T.B. • I.H.D.	• Date. • Site. • Outcome. • Blood transfusion.	Drugs of chronic use as: • Long acting penicillin (Rh. F). • Corticosteroid. • Contraceptive pills. • Anti T.B. • Anti hypertensive.

- ✓ Duration -----
- ✓ Manifested by -----
- ✓ Investigated by -----
- ✓ Treated by -----
- ✓ Complicated by-----

(5) Family History

- Consanguinity.
- Similar conditions.
- Chronic diseases: - DM / hypertension / TB / IHD.

If -ve
Irrelevant

(6) Socioeconomical state

- **High social class:-** Hypertension - I.H.D - irritable bowel disease
- **Low social class:-** malnutrition - infections.

(7) Menstrual and obstetric history

- Date of Menarche and menopause.
- Frequency of the periods, regularity, duration, amount of blood.
- History of intake of contraceptive pills.

Some side effects of contraceptive pills:

- Headache.
- Nausea and vomiting.
- Breast discomfort.
- Hepatic cholestasis, hepatic adenoma and Budd-Chiari syndrome.
- Thromboembolic manifestations.

GENERAL

Overview

A أرقام	1. Pulse
	2. Temperature
	3. Blood pressure
	4. Respiratory rate
B	5. B uilt
C	6. C olours
D	7. D ecubitus
E حاجة فوق وحاجة تحت وحاجة في النص	8. Neck vein – H & N (total)
	9. Clubbing (upper limb)
	10. Lower limb edema (lower limb)
F	11. Mentality "فكر"
	12. Face + general look

N.B.

Other systems: - More details

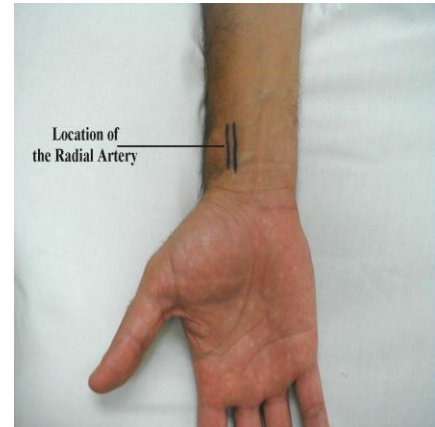
Other method for general examination

1. General condition
2. Mentality
3. Built
4. Decubitus
5. Face examination (colours / temperature / others)
6. Neck examination (neck vein / carotid artery / trachea / L.N. & thyroid)
7. U.L. examination:(pulse / B.P. / hand / L.N. & others)
8. L.L. examination (odema & peripheral pulsations)

(1) pulse

A pressure wave transmitted through arteries particularly if superficial & passed over a bone due to ventricular ejection.

Encircling the wrist & palpating the radial artery by the middle 3 fingers (index & ring fingers for slight compression & the middle Finger for palpation)



Comment on:

1. Rate
2. Rhythm
3. Volume
4. Equality (in volume) on both sides.
5. Special character.
6. Condition of the blood vessels
7. Peripheral pulsations.
8. Force.
9. Tension.

Rate

- A. **Normal HR:** 60 - 100 beat / min under complete physical & mental rest.
- B. **Tachycardia:** (Rate > 100 / min)
- C. **Bradycardia:** (Rate < 60 /m.)

N.B.

- If pulse is regular you may count in 30 sec. and multiply by 2 ,, or in 15 sec. and multiply by 4.

Rhythm

ده تعرفه قبل ال rate عشان تعرف تعد كويس ال heart rate

- A. Regular pulse:
- B. Irregular pulse:

i. Regular Irregularity:

- *Pulsus bigeminy or trigeminy:* dropped beat at regular rate

i. Irregular Irregularity:

1. A.F. (complete irregularity)
2. Multiple extrasystoles (occasional irregularity)
3. Ventricular fibrillation
4. Ht. Block with changeable degree of block.
5. A. flutter with changeable degree of A-V block.

	Extra systole (with S. tachycardia)	AF
Pulse		
Rhythm	Occasional irregularity	marked irregularity (I can't count 4 or 5 regular successive beats)
Rate	According to sinus	usually rapid e.g. 100- 150/min
Carotid massage		slowing of pulse
Exercise	May increase irregularity	increased irregularity
Pulse deficit	<10 beats/min	>10 beats/min
Res. sinus rhythm	Positive	negative
Neck veins		
A wave	Normal with Occasional irregularity	Absent
V wave		Systolic expansion
Heart sound		
	Normal with Occasional irregularity	Variability
ECG		
P	Premature beat followed by compensatory pause	Absent
QRS		markedly irregular

Volume

Volume: Pulse pressure (S - D) amplitude = 20 – 60 mmHg.

Judged by the movement of the palpating finger produced by the arrival of the pulse wave.

Big pulse pressure:

- Pulse pressure > 1/2 systolic blood pressure.
- Pulse pressure > diastole.

Small volume (small pulse pressure = small amplitude)	Big volume (Big pulse pressure = large amplitude)
• Small stroke v. (low COP) • Shock: Thready pulse; weak & rapid • Tachycardia	• A.I. - P.D.A. - AV fistula • Arteriosclerosis of the aorta • Bradycardia in C.H.B. • Hyperkinetic state

Hyperkinetic state:

H: - hypoxia – hyperthyroidism – Hepatic F. - hyperthermia

B: - Pregnancy – Beri Beri – Paget's

A: - Anemia - AV fistula – Anxiety

Causes of variable volume of pulse:

1. A.F.
2. V. Tachycardia.
3. 3rd H.B.
4. Pulsus alternans

Equality

Both radial arteries must be examined at the same time for equality as regard volume.

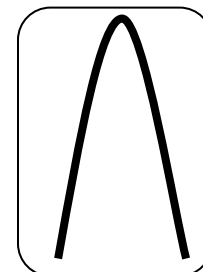
Causes of unequal pulse:

a) Compressing from outside	b) Causes in the arterial wall	c) Causes occluding the lumen
• Cervical rib. • Cervical L. Ns. • Pancoast tumour	• Aortic arch aneurysm • Coarctation of the aorta (pre ductal) • Aneurysm of subclavian a. • Arteritis.	• Thrombosis. • Embolism.

Special character**1- Water hammer pulse (collapsing pulse):**

Sharp ascending and sharp descending with high amplitude (e.g.: 160/50) less accurate pulse P. > 50

Causes of high pulse P. (see volume)

**2- Plateau pulse = Anacrotic pulse:-**

slow ascending and slow descending with low amplitude.

Causes:

- a. A.S. (moderate or severe cases)
- b. B- blockers therapy
- c. L.V.F.

**3- Pulsus paradoxus:**

Def.: Marked drop in the systolic pulse during inspiration (more than 10 mm Hg)

Causes:

1. Pericardial:- tamponade, constrictive or effusion
2. Severe congestive H.F.
3. COPD (especially severe asthma)

Detection: by sphygmomanometry

Measure B.P. during inspiration & during expiration.

4- Pulsus deficit:

HR on apex > radial pulse

Explain:

Contraction of an empty ventricle (so some weak beats are unable to reach the radial artery).

Causes:

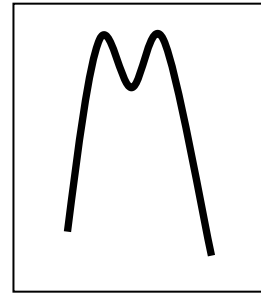
- A.F >10 beats/m
- Extrasystoles < 10 beats / m.

5- Pulsus bisferiens = bifid = double hump:-

Best seen & felt at carotid artery

Causes:

1. A.I. combined with A.S. (double A)
2. Severe A.I.
3. Hypertrophic obstructive cardiomyopathy

**6- Pulsus alternans**

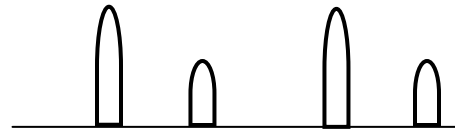
Alternation of strong & weak pulse waves with Equidistance, as volume is reduced in every other beat.

Causes:-

- Severe L.V.F.

Detection:

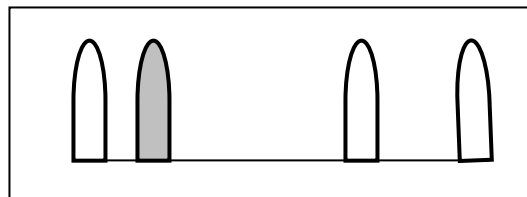
- by sphygmomanometer

**7- Pulsus bigeminy**

Strong beat followed by weak beat then compensatory pause

Causes:-

1. Ventricular premature beats
2. Digitalis toxicity
3. Myocarditis
4. Myocardial infarction

**8- Pulsus trigeminy, pulsus quadrigeminy****Causes:**

- multiple extra systole or
- Dropped beats.

Special Character of pulse associated with Aortic valve diseases:-

- A.I:- Water hammer pulse (Collapsing pulse)
- A.S:- plateau pulse
- A.I.+ A.S:- pulsus bisferiens

Vascular wall

- **Rolling maneuver**: - distal and proximal occlusion of small segment of that artery by the index and ring fingers, rolling of the middle finger.
- **Osler's maneuver**: - occlude the brachial a. by one hand (or by sphygmomanometer cuff) & palpate the radial a. by the other hand.

Result:

Normally the arterial wall is not felt (or felt & elastic)

Causes of palpable arterial wall:

- Systemic atherosclerosis.
- Focal arteriosclerosis.
- Polyarteritis nodosa: grape like along course of the artery.

Peripheral vascular

Other peripheral arterial pulsations & capillary pulsation

Radial	Lateral to the tendon of flexor carpiradialis.
Brachial	At the elbow medial to the biceps tendon.
Subclavian	Pressing downward above the middle of clavicle.
Carotid	Medial to the sternomastoid muscle At the level of cricoid cartilage Against the transverse process of 6th C. vertebrae .
Femoral	At the patient midpoint of inguinal ligament while the thigh is flexed and abducted.
Popliteal	Middle of the popliteal fossa while the patient lies supine with the knee slightly flexed.
Posterior tibial	Behind the medial malleolus.
Dorsalis pedis	Lateral to the extensor hallucis longus tendon against navicular bone on the dorsum of the foot. In the 1st interossus space (normally absent in 15% of population).

Other findings not involved in usual examination:

1. **Femoral-radial pulse relationship** : the femoral pulse is weaker & delayed when compared with radial pulse simultaneously in:
 - Coarctation of A.
 - Saddle embolus at the bifurcation of the aorta.
2. **Unequal carotid pulsations** :-
 - Atheroma of innominate a. or common carotid a.
 - Aortic Aneurysm or dissecting Aneurysm.
3. **Weak or absent dorsalis pedis pulse**:
 - As causes of unequal pulse

Example:-

The patient's pulse is 75/min., regular, average volume, equal in both sides, no special character, the condition of the blood vessels are normal with palpable pulsations of dorsalis pedis artery.

(2) Temperature**Measurement:**

1. **Oral temp.:** Under the tongue with closed lips for 3 min

N= 36.5 – 37.2 °C.

False decrease:

- a. Mouth breathing.
 - b. Incomplete closure of mouth.
 - c. Putting thermometer for too short time.
2. **Rectal temp.:** Left for 2 min. (0.5 °C higher).

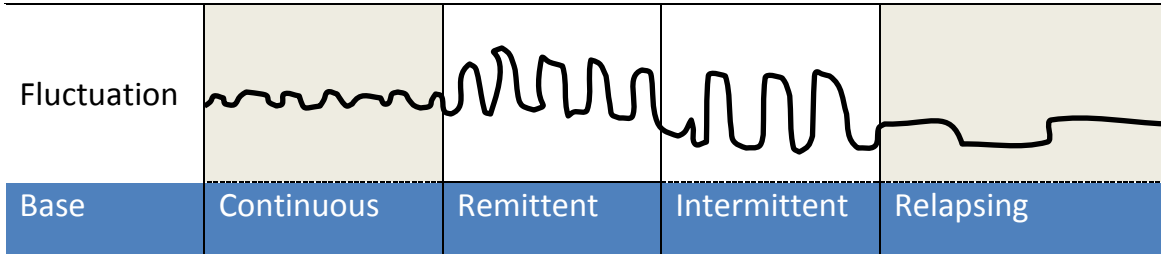
Indicated in:

- Infants.
 - Irritable pts.
 - Painful oral lesions.
 - Insane.
 - Comatosed pt.
3. **Axillary temp.:** Put in axilla for 3 min. (1/2 °C Lower).



Terms:

1. Subnormal Temp. $< 36.5^{\circ}\text{C}$.
2. Fever $> 37.2^{\circ}\text{C}$.
3. Hypothermia $= \leq 35^{\circ}\text{C}$.
4. Hyperthermia $= > 41^{\circ}\text{C}$.

Types of fever:

1. **Continuous:** (Gram negative sepsis, CNS damage)
Fluctuation: $< 1^{\circ}\text{C}$ & $>$ base line.
2. **Remittent:** (Viral, TB)
Fluctuation: $> 1^{\circ}\text{C}$ & $>$ base line.
3. **Intermittent - Hectic:** (Abscess- malaria)
Fluctuation: $> 1^{\circ}\text{C}$ & may reach the base line.
4. **Relapsing (cyclic, periodic):** (Collagenosis, lymphoma, brucellosis, IMN & FMF)
Days of fever and days of normal

Temperature – Pulse relationship**a) Synchronization between rise in body temperature and pulse rate documented as:**

Each $\uparrow 1^{\circ}\text{C} \Rightarrow \uparrow 10 - 15$ beats/m in HR

b) Asynchronous form:

Relative Tachycardia	Relative bradycardia
HR more than expected for body temperature as Myocarditis (Rh. F/Diphtheria) .	HR less than expected for body temperature as typhoid F/ meningitis .

(3) Blood pressure



مبدئيا كده ثبت الصوره دي في ذهنك
لانها الصح ومتعملش غيره ا

Factors affecting blood pressure:

Systolic	Diastolic
1. C.O.P (Lt. V. systolic P. & stroke volume).	1. Peripheral resistance (vascular tone) & intact aortic valve. 2. Elasticity of Aorta. 3. Bl. Volume. 4. Bl. Viscosity.

Normal Blood Pressure in adults:

- **Systolic:** 100 – 140mm Hg.
- **Diastolic:** 60 – 90mm Hg.

Less in children & young adults & more in elderly (in systolic P)

Hypertension: in adult; suggested when:

- **Systolic:** over 140mm Hg.
- **Diastolic:** over 90mm Hg.

At least twice in 2 separate occasions under complete mental and physical rest.

New classification of blood pressure measurements

Category	Systolic BP	Diastolic BP
Optimal	< 120	< 80
Normal	<130	< 85
High normal	130-139	85 – 89
Hypertension		
mild (Stage I)	140-159	90 – 99
Moderate (Stage II)	160-179	100 – 109
Severe(Stage III)	> = 180	> = 110
Isolated Systolic Hypertension		
grade I	> 140-159	< 90
grade II	> =160	< 90

Measurement of B. P.:

- **Direct:** direct intra-arterial measurement.
- **Indirect:** by sphygmomanometer (palpatory, Auscultatory)

Palpatory:

- **Advantage:** avoidance of auscultatory gap.
- **Disadvantage:** can't detect diastolic B.P.

auscultatory:

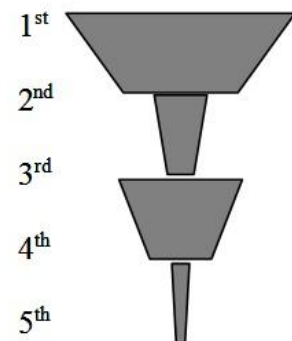
- **Advantage:** disadvantage of palp.
- **Disadvantage:** advantage of palp.

Kortakoff phases.

1. 1st sound heard.
2. Sounds markedly decreased or may disappear (mistaken for D).
3. Sounds reappear (mistaken for S).
4. Sudden muffling of the sounds.
5. Total disappearance of the sounds.

Some measurement:

- Rt. & Lt. Sides: equal; difference normally is < 10mm Hg.
- UL & LL.: Normally LL > UL. By 10 - 20 mm Hg.
- During Standing & during lying: for postural hypotension.



Technique of measurement of B. P.

Patient

1. Position the patient's arm so the anticubital fold is of level with the heart.
2. Make sure that they do not have any tight clothing which may constrict their arm
3. Under complete mental and physical rest



Apply blood pressure cuff

1. The bladder of the cuff should encircle 80% of the arm / not tight nor loose.
2. The centre of the cuff bladder should be placed over the line of the brachial artery.
3. Approximately 2 cm above the anticubital fold.

The doctor

1. Palpate the patient's radial pulse and inflate the cuff until you feel the exact point when the pulse disappears (estimated systolic pressure).
2. Place the stethoscope over the brachial artery and inflate the cuff 30 mmHg above the estimated systolic pressure.
3. Release the pressure slowly, no greater than 5 mmHg per second.
 - The level at which you consistently hear beats is the systolic pressure.
 - Continue to lower the pressure until the sounds disappear. This is the diastolic pressure.



Lastly

Remove the cuff and thank the patient.

Important findings:

- Blood pressure in lower limbs > blood pressure in upper limbs by less than 20 mmHg which is normal and called "Hills phenomena" due to:
 - ✓ True increase: as arteries in lower limbs are in line with aorta.
 - ✓ False increase: as arteries in lower limbs are surrounded by bulky muscles,, so muscle need high pressure in bag to occlude the artery.
- If blood pressure in lower limb > blood pressure in upper limb by > 60 mmHg it is called: "**Hills sign**" in AR.
- If blood pressure in lower limb < blood pressure in upper limb it is called: "**reversal of Hills**" and occur in co-arctation of aorta.
- **In postural hypotension (orthostatic hypotension = orthostatic syncope)**

During Standing there is large fall in both systolic & diastolic B.P. (Normally there is a slight fall in systolic B. P. < 20 mm Hg and increase in diastolic B. P. < 10mmHg).

- **B.P. in obese arm :**
 1. Use wider cuff that can encircle the arm completely, or
 2. The cuff wrapped around the upper 1/3 of the forearm & diaphragm of stethoscope is positioned over the radial artery.
- **P. In the LL**
 1. Pt. is lying in prone position.
 2. Apply the cuff around the lower 1/3 of the thigh above popliteal fossa.
 3. Auscultate (or palpate) popliteal artery.
 4. Continues as in U.L.

Uses of sphygmomanometer

1. B.P. & pulse changes:

- a) Measurement of B.P.
- b) Determination of pulse pressure (S—D)
- c) Detection of pulsus alternans: if pressure is between weak beat & strong beat the radial pulse will drop by half as, the weak beats will be abolished.
- d) Detection of **pulsus paradoxus**: systolic B. P. will drop during inspiration (more than 10 mm Hg.)
- e) Detection of inequality of pulse in both sides (different blood pressure in Rt. & Lt limb).

2. In some valvular disorders:

- a) **In coarctation of aorta**: B.P. in arms more than B.P. in legs by more than 60 mm Hg (reversal of Hill's).
- b) **In A.I.** : B.P. in leg is more than B.P. in arm by more than 60 mm by (Hill's sign)

3. Diagnostic tests:

- a) Capillary **fragility** test (Hess test)
- b) **DVT**: application of low pressure will cause severe pain in diseased leg
- c) Diagnosis of tetany: **Trousseau's** test (carpo- pedal spasm).
- d) **Walker's** test for diagnosis of Myasthenia gravis.

4. Others:

- a) Tourniquet in venesection.
- b) Haemostasis.

Auscultatory gap – silent gap

Definition period during recording of the blood pressure by auscultation in which no sounds can be heard but blood pass and can be felt.

N.B.

- Present in some hypertensive patients.
- Mechanism unknown.

Importance: false diagnosis of systole or diastole.

how to avoid:

- Do palpatory method at first to know roughly systolic blood pressure.
- Rise pressure in the bag 20 mmHg above systolic blood pressure known by palpation.
- Do auscultatory method.

(4) Respiratory rate

See chest

(5) Built

Determined by noting the **weight** and **height** in relation to:

- Age
- Sex.

(A) Height

Dwarf (stunted growth): as in

1. Cong. cyanotic H. Diseases:- F4
2. Prolonged H. dis. since childhood.

Tall: as in

Marfan's syndrome (tall, arachnodactyly, pes cavus, high arched palate, ectopic lens, up ward subluxation of lens and A.I.)

(B) Weight

It can be determined by **body mass index (BMI)** which is derived from the formula

$$Wt (Kg) / Ht (m^2)$$

- **Normally** it is "18-25" (average body built).
- **Under built:** < 18.
- **Overbuilt:** 25-29.
- **Obesity:** 30-39.
- **Morbid obesity:** 40 or more.

Obesity can be assessed by the thickness of skin folds e.g.,

- **Lateral aspect of the arm** 0.9 – 1.1 cm.
- **Abdomen** = 1.5 cm.
- **Buttocks** = 1.5-2.5 cm.

Waist / hip ratio (in male <0.95 in female <0.85)

- Apple shaped persons with a greater waist-hip ratio have an increased risk of cardiovascular disease

Feature**1. The height and span are almost equal.**

- Height:- distance from the occiput to the heels in upright position.
- Span:- distance between the tip of the fingers with outstretched hands.

2. Lower and upper segments are usually equal

- L. segment = distance between symphysis pubis and floor
- U. segment = distance between occiput and symphysis pubis

(6) Complexion (colors)

- a. Pallor
- b. Jaundice
- c. Cyanosis
- d. Pigmentation
- e. Skin rashes

*Pallor***Site of examination:**

1. Inner aspect of lips.
2. Skin of the face.
3. Nails.
4. Palm creases.

Never in Conjunctiva because of endemic trachoma in Egypt

N.B.

- The degree of pallor depends on the state of capillaries, amount of blood within the capillaries, Hb, pigmentation & thickness of the skin.
- Examination of the mucous membranes may help to distinguish pallor of anemia from that of other causes.

Q: Causes of pallor?

- Anemia.
- Shock or ↓COP.
- Toxemia e.g. infective endocarditis.
- Edema of the face e.g. nephrotic syndrome.
- Racial pallor (Far East).

Q: Causes of pallor with normal CBC?

Jaundice

Def. yellow discoloration of skin & mucous membranes due to increase level of bilirubin > **2.5 mg %**.

Sub clinical Jaundice: - Serum bilirubin = 1- 2.5 mg/dl.

Site of examination: In sclera of lower fornix in the day light.



A. **Hepatocellular J** :- e.g.:- Viral hepatitis, cirrhosis:

Orange yellow $\pm$ L.C. F. Manifestations Liver is enlarged & tender, later it may become firm and shrunken.

B. **Hemolytic J**.: Lemon yellow J. $\pm$ pallor

C. **Obstruction J**.: Olive green J. + pruritus, bradycardia & frothy urine.

DD of yellow discoloration of skin & sclera:

1. Atebrin
2. Hypercarotenaemia (not in sclera)
3. Picric a. toxicity
4. Uraemia
5. Myxoedema
6. xanthomatosis.

Cyanosis

- **Def. & pathogenesis:** it is bluish discolouration of skin & m.ms, due to presence of more than 5 gm/dl reduced Hb.
 - ✓ Cyanosis is aggravated with polycythaemia.
- **Site of exam:** tongue, lips, hands; nails.
 - ✓ Examination in daylight is essential.
- Types:
 - ✓ Central.
 - ✓ Peripheral.

Don't forget

- In jaundice level of bilirubin > **2.5 mg %**.
- In cyanosis presence of more than **5 gram / dl** reduced Hb.

Central cyanosis		Peripheral cyanosis	
Blood ejected from heart (or lung) already contains more than 5gm% reduced Hb (hypoxic hypoxia)		Blood ejected from heart is normal (normal amount of reduced Hb (stagnant hypoxia))	
Causes			
A- Cyanotic lung disease : 1) Asphyxia. 2) High altitude. 3) Pulmonary A-V fistula. 4) Lung diseases:- COPD, fibrosis, collapse, Massive consolidation, Pneumothorax or Pulmonary embolism		A- Generalized decreased blood Flow : 1- Polycythaemia: increased viscosity. 2- Marked decrease in C.O.P. 3- systemic venous congestion : (RVF& cardiac Tamponade) 4- shock	
B- Congenital cyanotic H. dis. With Rt. To Lt. Shunt : 1- Fallot's tetralogy 2- Eisenminger's		B- Localized decrease in blood flow : 1 - Cold temp. 2- Peripheral circulation disturbance (Raynaud's, Burger's, venous thrombosis & Arterial occlusion)	
C- Ploycythaemia.			
D- False (chemical) cyanosis: Met-Hbaemia: cong. / nitrites Sulph-Hbaemia: sulpha or bacteria			
examination			
	Central cyanosis	Peripheral cyanosis	
Site	All the body: Skin, conjunctiva, inner lips & tongue (and as in peripheral) (Tongue is cyanosed)??	Skin of peripheral parts: Tip of fingers, hands, tip of nose, ears, outer lips (Tongue is normal)	
Hand	Warm (peripheral V.D.)	Cold (peripheral V.C.)	
Warming	No effect	Cyanosis improved	
Oxygen	Improves cyanosis (in pulmonary causes only)	No effect	
PO2	Decreased arterial and venocapillary blood	Decreased in venocapillary but normal in arterial blood	
Clubbing	±	-ve	
Ploycythaemia	+	-	

Why Tongue?

1. Has no autonomic fibers
2. Highly vascular
3. Continuous exercise
4. Warm site

Differential central cyanosis: in the lower half of the body only

- PDA with reversed shunt.
- PDA with coarctation of aorta.

Pigmentation

Causes of general pigmentation	Localized pigmentation
1. Familial / Racial. 2. LCF. 3. RF. 4. Addison's. 5. Pellagra. 6. Haemochromatosis.	As butterfly area of the face • Red → molar rash (MS) • Discoid → SLE • Brown → pellagra • In pregnancy (chloasma gravidarum)

(7) Decubitus

Position of the patient in bed in relation to certain disease.

☞ **Orthopnea:** - Semi setting position

1. *Heart:- Lt. Sided H.F. & Pericardial effusion.*
2. *Chest:- Br. Asthma & Emphysema*
3. *Increased intra abdominal pressure e.g. massive ascites.*
4. *Marked obesity.*

☞ **Squatting position:** In Fallot's tetralogy.

☞ **The praying Muslim position:** pericardial effusion and mediastinal syndrome.

☞ **Lateral Position in chest diseases:** pleurisy, lung abscess or pneumonia.

☞ **Trepopnea:** dyspnea on healthy side in sever unilateral lung collapse.

☞ **Opithotonus:** (high arched back) meningitis, tetanus or strychnine poisoning.

☞ **Dorsal** (flexion)

- **Passive** in abdominal colic.
- **Or Rigid** in peritonitis.

☞ **Platypnea** dyspnea in the erect position relieved by recumbency In huge apical lung tumours or hepatopulmonary.

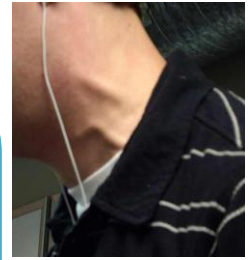
(8) Neck vein

Value: - reflects pressure changes inside the right atrium.

- The venous pressure is measured most accurately by manometry.
- Internal jugular vein is preferred:-

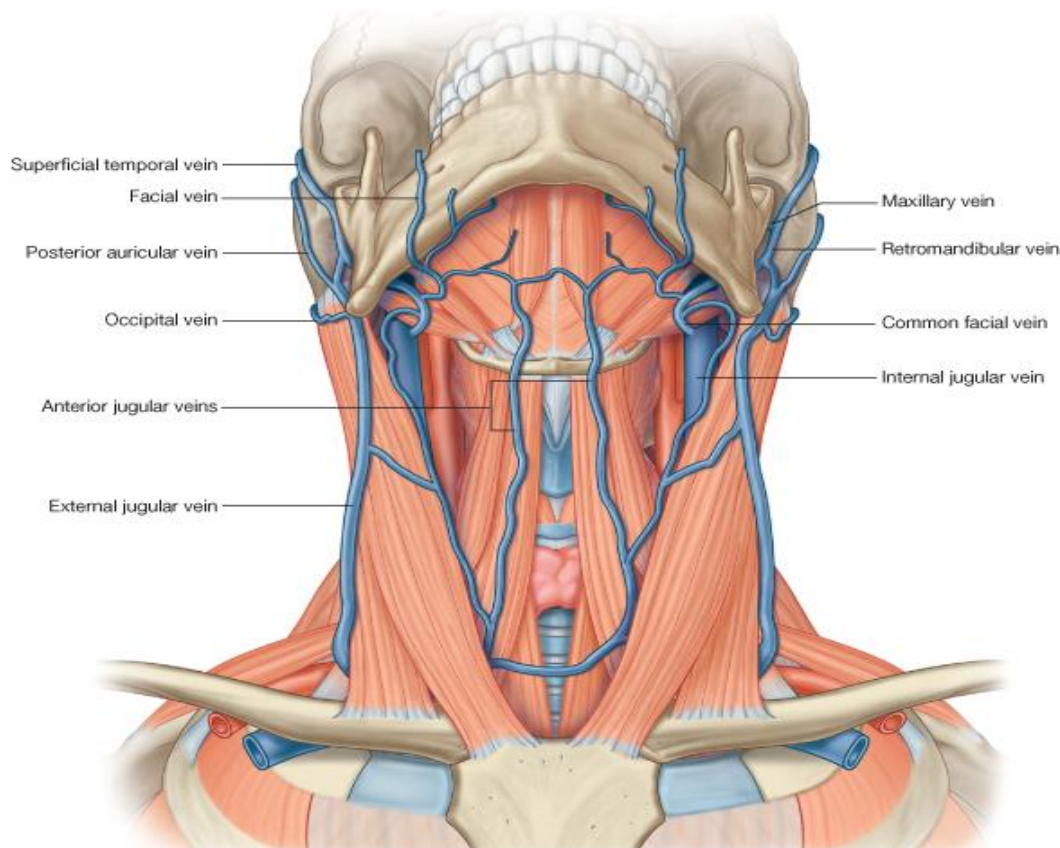
Internal jugular vein	External jugular vein
Valvless	Has a very variable course & may be congenitally absent.
in direct continuity with the Rt. A.	may be obstructed by: a. Its tortuous course. b. muscular layers through which it passes

- **Right sided pulsations are preferred**:- Lt. Innominate vein (= brachiocephalic) may be compressed by the arch of the aorta.



Examine Rt. Internal jugular vein:

- **Surface anatomy of Internal jugular vein:** Straight line from the angle of the jaw to the medial end of the clavicle deep to sternomastoid & platysma muscles.
- **External jugular vein:** is only informative if pulsating.



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It is venous or arterial ???

	Venous	Arterial
Sternomastoid	Lower Lateral (L.L.) to sternomastoid in post. Triangle.	Medial & upper to sternomastoid in ant. Triangle.
Ch.Ch.	Better seen than felt	Better felt than seen
	Wavy: more than one wave Have an upper level	One wave Have no upper level
Disappear	Disappear by pressure Disappear by deep inspiration	No
Relation	Postural changes Hepato-jugular (one minute abdominal compression test)	No

Comment on neck veins:-

- A) Waves (J.V. pulse)
- B) Pressure (column elevation = congestion)

N.B.

To know the relation between neck veins and cardiac cycle you have to remember at first cardiac cycle



1. Iso metric contraction phase.
2. Ejection phase.

1. Iso metric relaxation phase.
2. Passive filling 70%.
3. Atrial contraction 30 %.

wave

Physiology

- Consists of three positives (A, C and V) and two negatives (X and Y).
- The A wave is normally the highest wave.
- The X wave is usually deeper than the Y wave.

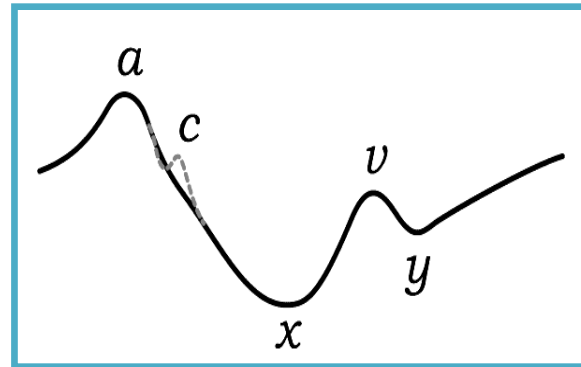
- Radial pulsation is preferred for timing of the venous pulsations.

Wave	Explained	Timing
A Wave:-	Atrial contraction.	Presystolic
C Wave :-	- Elevation of the tricuspid valve at the start of ventricular contraction - Transmitted pulsation of carotid artery.	
X (Systolic collapse)	Right atrial relaxation.	Systolic
V Wave :-	Accumulation of venous blood in the right atrium during ventricular systole.	Systolic
Y descent: - (70 %)	Descent of blood from right atrium to right ventricle (diastolic collapse)	Diastolic

Abnormalities of the waves:

A wave

- Large or giant A waves:**
T.S. / P.S. / PH++
- Absent A:** - Atrial fibrillation.
- Canon A wave:-** Nodular rhythm / V. tachycardia / 3rd HB



Prominent V wave (systolic expansion)

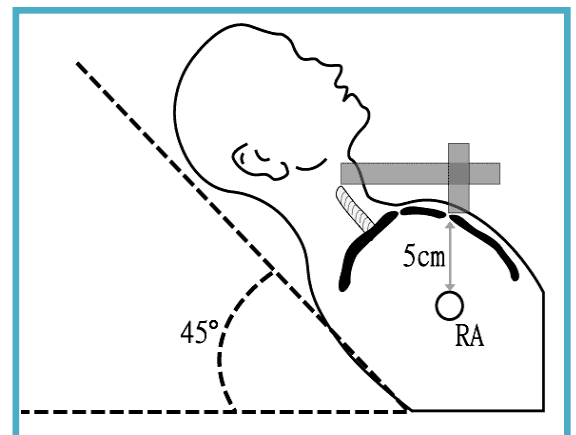
- T.R.
- Right sided heart failure
- AF.
- Canon waves

Pressure

Definition: - it is the vertical height between the top of venous pulse & angle of Louis when the Pt. is lying at an angle of 45°.

Measurement:

- The Pt. is positioned at about 45° to the horizontal plane.
- The head is supported by a pillow & the neck is slightly flexed to allow the skin & muscles overlying the vein to relax.
- The jugular V. pressure is measured as "The vertical



distance bet. the manubrio-sternal angle & the top of the venous column

Normal jugular venous pressure:

- The angle of Louis is about 5cm above Rt. A. at 45.

$$\text{C.V.P.} = \text{J.V. pressure [----]} + 5\text{cm} = \text{---- cm H}_2\text{O}.$$

- Normal jugular venous pressure not more than 2 cm H₂O.
- Normal pressure in Rt. A. (central venous pressure) less than 7 H₂O

Congested neck veins:

A. Congested pulsating neck veins:

- *Right sided heart failure.*
- *Tricuspid valve disease as T.I. & T.S.*
- *Pericardial (effusion - Constrictive pericarditis).*
- *Increased intrathoracic pressure (Massive pleural effusion \ Tension Pneumothorax / Emphysema).*
- *Increased intra-abdominal pressure (Tense ascites / Pregnancy / Huge abdominal swelling).*
- *Over transfusion = hypervolaemia*
- *Hyperdynamic circulation.*

B. Congested non pulsating neck veins:

- *S.V.C. thrombosis causing complete obstruction.*
- *Mediastinal syndrome causing complete obstruction.*
- *Severe late constrictive pericarditis & pericardial effusion*

N.B.

1. Any severe congestion may appear as congested non pulsating.

2. Neck vein & respiration:

- Insp. → emptying (normal).
- Insp. → no change
 - ✓ Congestive H.F. or,
 - ✓ S.V.C. obstruction.
- Insp. → filling in pericardial disease "Kausmaul's sign".
- Exp. → filling in emphysema.

(9) Clubbing

Def. & pathology: Proliferation of soft tissues at nail beds & terminal phalanges



Causes of clubbing:

	(I) Pale (toxaemic)	(II) Blue (hypoxic / cyanotic)
Cardiac	Infective endocarditis.	• cong. cyanotic heart: Fallot's / Eisenminger's • Corpulmonale (due to underlying lung diseases)
Chest	• Suppurative lung S. • Bronchogenic carcinoma • Mesothelioma.	Cyanotic lung dis.: • Fibrosing alveolitis • any sever lung dis (resp. F).
GIT	• B polyposis • Cirrhosis especially biliary Ulcerative colitis. • Steatorrhoea	

COPD (if associated with bronchiectasis)

(III) Familial

(IV) Occupational: some fingers e.g. index & thumb (shoemakers).

(V) Unilateral clubbing: causes of inequality of pulse on both sides,

(VI) Differential clubbing (in L.L.S only): causes of differential cyanosis.

Causes of reversible clubbing:

(Empyema, mesothelioma & endocarditis).

Grades of clubbing:

1. **Grade I:** obliteration of the angle between nail and nail bed (+ ve fluctuation test at nail base).
2. **Grade II** (parrot beak): I + increase convexity of nails in its longitudinal curve.
3. **Grade III** (Drum stick): I + II + hypertrophy of terminal phalanx.
4. **Grade IV (hypertrophic osteoarthropathy):** III + "tender" Hypertrophy of distal ends of long bones at wrist & ankle due to periosteal irritation & new bone formation.



Exam.

1. Look at fingers in profiles.
2. Window test (Schamroth's window test: pt's holds 2 index finger nails touching each together: if normal, will show a diamond-shaped window).
3. Fluctuation test at the nail base.

**N.B.****Causes of unilateral clubbing in one upper limb**

- Thoracic inlet syndrome (cervical rib – pancost tumor).
- Aortic aneurysm, arteriovenous fistula in dialysis patients.

(10) Oedema

Definition: means abnormal accumulation of fluid in the interstitial tissue due to disturbed mechanisms of formation of interstitial fluid.

Comment on:

1. Unilateral or bilateral
2. Pitting or non pitting
3. level (extent)
4. Tenderness.
5. Examination of serous membranes.

Examination of oedema:

1. **Below the knee** pressure for about ½ minute over bony prominence (Posterior to medial malleolus, dorsum of the foot and the chin of tibia)
2. **Pinching** test over the thigh
3. **Pedou-orange** over the anterior abdominal wall.
4. **Examine back** for sacral edema.

D.D. of generalized edema**A- Cardiac oedema:** with congested neck veins.

- Bilateral.
- Painless.
- Pitting.
- Dependant.
- Before ascites.

Dependent: appears in the dependant parts of the body:

- Ankle oedema: in ambulant Pt.
- Sacral oedema: in recumbent (or bed ridden) Pt. (When oedema is very great; it may affect whole LL, genitalia, abdomen, chest and even the face "**generalized anasarca**")

Pitting

Before ascites: always L.L oedema precedes ascites except in 2 conditions where ascites precedes (ascites precox):

- pericardial effusion & constrictive pericarditis.
- T.R. or T.S. due to marked congestion of liver

Bilateral

- Unequal oedema occurs in case of deep venous thrombosis (DVT).
- It may be on one side in Pts. preferring to sleep on one side.

Q : Pathogenesis of cardiac oedema ?**B- Renal oedema:**

Occurs firstly in eye lids, generalized, pitting marked in the morning.

A-Nephritic: in acute nephritis	B- Nephrotic:
- Oliguria - Hypertension (Na & water ret) - Haematuria - Epith & red casts in urine	- Heavy albuminuria - Hypoproteinaemia - Generalized oedema - increased cholesterol

C- Hepatic oedema:

In advanced liver cirrhosis + other signs of L.C.F.

D- Nutritional oedema: + other signs of nutritional deficiencies.

- Sever malnutrition.
- Sever malabsorption.
- Sever anaemia.

E- Angioneurotic oedema: allergic.

- Non pitting – Acute - Localized (lips, eye lids, larynx) or generalized
- History of other allergic manifestation: eczema., asthma.
- Family history of allergy.
- Rapid response to anti allergic measures.

Localized edema

- D.V.T.**
- Lymphedema** :(non pitting)
Filariasis - Post mastectomy.
- Varicose veins**
Mainly in L.L.+ signs of varicosities.

4. **Orthostatic edema:**

Mainly in L.L + diurnal variation ± Occupational Factors

5. **Angioneurotic edema**

Affecting the face (lips) asymmetrically, with history of allergy. Of sudden onset, self limited

N.B.

1. **Causes of edema in one UL or one L.L.:** DVT, cellulitis, lymphedema trauma
2. **Drugs causing oedema** e.g. cortisone, pills, NSAID.
3. **Non pitting edema** occurs in Lymphedema, myxoedema and Angioneurotic edema.
4. **Occult Oedema:** Up to 3 liters of excess fluid may be retained in the interstitial tissue without apparent oedema Pitting oedema is usually demonstrated if accumulating exceeds this amount (3 liters).

(11) Mentality

Consciousness, orientation, mood (= emotional state), memory intelligence and behavior.

Example: The patient is fully conscious, oriented with time, place & persons, emotionally stable of intact memory as regard near & far events of average intelligence & behaves normally.

(12) Facial expression

General look & Face

A. General condition

- Good.
- Bad
- Fair.
- Cachetic appearance in advanced Malignancies.
- Infantile appearance as infantilism.

B. Face (see below)**Upper limb**

1. **Appearance and size of hand and fingers:** e.g. spade hand (Acromegaly)
2. **Temperature of hands.**

a) Warm hands:	b) Cold hands:
• Fever • Hyperdynamic circ • Central cyanosis.	• Low C.O states. • Peripheral cyanosis. • Neurotic encephalitis.

3. Sweating

- ✓ Thyrotoxicosis.
- ✓ Toxaemia.
- ✓ Neurosis.
- ✓ Hyperhydrosis

4. Capillary refill**5. Osler's nodes / Jan way's patches / Splinter Hemorrhage** (Inf. End)**6. S.C. nodules:** - S.C. firm non tender nodules on extensor surface of arm & along tendons.(Rh. Fever / Rheumatoid arthritis).**7. Nails:** (see above)**8. Joint abnormalities:** - (as Rh. A.)**9. Edema:** (as above)**10. Lymph Nodes of the Upper Extremity:**

- a. **Epitrochlear Nodes:** in the inner aspect, just above the elbow.
- b. **Axillary Nodes:**
 - Ask the patient to lift both arms away from the sides of his body.
 - Then extend the fingers of both your hands and gently direct them towards the apices of the arm pits.

Describe the following

- **Size:** Pathologic nodes are generally greater than 1 cm
- **Firmness:** Malignancy makes nodes feel harder
- **Quantity:** The greater the number of nodes, the more likely true pathology exists
- **Pain:** Often associated with inflammation (e.g. infection)
- **Relation** to other nodes and surrounding tissue

Signs of nails examination

1. **Colour changes**

- **Pallor**: see before.
- **Cyanosis**: see before.
- **Leuconychia**: Hypoalbuminemia
- **Yellow**: Nicotine stains/Yellow nail syndrome [peripheral edema, bronchiectasis, pleural effusion]
- **Red Cherry red**: CO poisoning
- **Terry's nails** (distal half is brown, while proximal half is white-pink): Cirrhosis / Chronic renal failure
- **Nail bed erythema = telangiectasia**: SLE

2. **Spooning (Koilonychia)**: in sever chronic

- iron deficiency anaemia.

3. **Capillary pulsation**: in A.I.

4. **Splinter Hges**: linear hges under nails.

Causes:

- Trauma in manual workers (common)
- Infective endocarditis.



5. **Onycholysis**: Trauma/ Psoriasis/Thyrotoxicosis/ fungal infection.

6. **Bands**

Beau's: multiple, unpigmented, transverse lines (Shock / Malnutrition / Weight loss)

Mees's :- solitary, white, transverse band (Renal failure / Chemotherapy)

Muehrke's :- multiple, opaque, transverse bands (Hypoalbuminemia / Chemotherapy)

Lower limb examination

1. **Oedema**: (see above)
2. **Cyanosis**.
3. **Clubbing of toes**.
4. **Pellargic rash** over the greater trochanter.
5. **B.P. difference between upper and lower limbs** (Hill's and reversal of Hill's)
6. **Venous system** for D.V.T & Varicose veins.
7. **Deformity** as in: Marfan's, Duchenne, myopathy, Friedrieich's ataxia & other congenital diseases.

Face

Parkinsonism	Mask like face
Myxoedema	Apathetic look
Hyperthyroidism	Restless, staring look
Acromegaly	Ape like appearance
Cushing's	Moon face
Congenital syphilis	Square like bulldog
Uremia	Earthy look
Myasthenia gravis	Weak smile / bilateral ptosis / snarl
Facial palsy	See neuro
Horner's syndrome	See neuro
Myopathic face	Expressionless, protruded lower lip
Toxic look	Infective endocarditis
Mongoloid face	Thalassaemia / Down
Mitral face: (malar flush)	peripheral cyanosis of cheeks (low C.O). Dilated capillaries. lips may be cyanosed.
Tricuspid face	tinge of jaundice + peripheral cyanosis of the lips.

Head & Neck

skull

- **Shape:-** Dolichocephaly , oxycephaly, or Boxy head (rachitic head)
- **Depressed fractures.**
- **Hydrocephalus** and Paget's.
- **Microcephaly** and **craniostenosis**.
- Enlarged supraorbital ridges :- **acromegally** (forntal sinuses)
- Tender temporal artery :- **Giant cell arteritis** .
- Bruit on :- intracranial **arteriovenous malformation**.

Eye

1. **Eye** **eyebrow** :- Loss of hair in outer 1/3 of eyebrow:
 - ✓ Myxodema.
 - ✓ Leprosy.
 - ✓ Artificial.

2. Eye ball

Exophthalmos	Enophthalmos (sunken eye)
• Thyrotoxicosis • cavernous sinus (pulsetile) • leukaemic deposits behind eye ball • Congenital	• Dehydration • Horner's syndrome

3. Eyelids

Ptosis	Oedema and puffy eye lids
• Congenital. • Hysterical. • Mechanical. • Oculomotor nerve paralysis • Myasthenia gravis • Ectropion. Entropion • Blepharospasm	• Renal, nephrotic or nephritic • Myxoedema . • Lack of sleep or excessive sleep • Chronic cough. • Angioneurotic • Rarely as a part of generalized oedema (HF / LCF)

4. Sclera

Blue sclera:

• Ankylostoma anemia • Congenital glaucoma • Children	• T . B. • Osteogenesis imperfecta (V imp) • Healthy persons
---	---

Jaundice : See complexion

5. Conjunctiva

a. Pallor :- conjunctiva is not reliable to diagnose pallor.

b. Sub conjunctival hemorrhage: - as in

- ✓ blood disease
- ✓ trauma
- ✓ cough
- ✓ server hypertension
- ✓ Infective endocarditis

It usually has an upper limit to be differentiated from conjunctival congestion.

c. Conjunctivitis: - C\P(photophobia, lacrimation and a sticky discharge).

d. Vitamin deficiency : as Xerosis (Vit .A), Vascularization (Vit.B2) Bitots

e. **Pingueculae** are triangular yellow deposits beneath the conjunctiva between the canthus and the edge of cornea, they develop with advancing years, and are of no clinical value.

f. **Pterygium** patch of progressive fibrosis may encroach upon the cornea.

6. **Cornea**

- **Arcus senilis** in senile patients and young with hyperlipidaemia
- **Opacitis** due to trauma or infection.
- **Kayser Fleisher ring** Wilson's disease.

7. **pupil**

- **Irregular**: - Argyll Robertson pupil
- **Miotic**: - Horner's syndrome
- **Mydriatic**: - 3rd cr. N. paralysis / opiate / atropin

8. **Lens :**

a. Cataract occurs in:

- Diabetes mellitus .
- Cretinism.
- Mongolism
- Scleroderma.
- Myotonia atrophica.
- Hyperparathyroidism

Rubella: stigmas indicating maternal rubella infection:

- Cataract -Mental defect -Nerve deafness
- Associated with cardiac (PDA or P.S.)

b. **Ectopic lens** : in Marfan's syndrome

9. **Iris and ocular tension**

- **Iritis** is often a manifestation of systemic disease e.g. ankylosing spondylitis and Behcet's disease.
- **The ocular tension** can be tested digitally, it is tested in patients with headache and diminished visual acuity.

10. **Fundus examination:**

- a. **Optic neuritis and its causes.**
- b. **Papilledema and its cause.**

c. Diabetes M.**d. Hypertensive retinopathy :**

- ✓ Grade I : mild sclerosis (narrowing) of retinal arteries .
- ✓ Grade II: Moderate to marked sclerosis with compression of veins at crossing
- ✓ Grade III : flame like hges + fluffy cotton exudates
- ✓ Grade IV : Papilledema + as in III

e. Infective endocaditis .

- ✓ Central retinal art. Occlusion by embolism.
- ✓ Central spots: (due to vasculitis).

f. SLE :- hard exudates = cytooid bodies / hges.**g.** Capillary pulsations in A.R.

11. **Xanthelasma** : It is a yellow eruption at inner side of the eyelids and peri-orbital skin associated with hypercholesterolaemia.

Nose and ear

- Redness of the tip of nose : alcoholism .
- Depressed nasal bridge (saddle nose) :- congenital S, traumatic or congenital or Wegner's granuloma.
- Working ala nasi: - pneumonia or nervousness.
- Sulfur granules: - B2 deficiency.
- Large nose: - congenital - acromegaly
- gouty tophi on helix, discharge chalky materials
- F.B in the external meatus
- Spider nevi
- Large ears: congenital - acromegaly - myxoedema.

Cheeks (See localized pigmentation)

- Pale :- anemia
- Red:- S.L.E & Cushing disease.
- Malar flush: - M.S.
- Brownish pigmentation: - pellagra.

Lips

- **Cheilosis** due to riboflavine deficiency causing denuded epithelium at the line of colour of the lips, peeling toward the mucocutaneous junction.

- **Angular stomatitis** (angular cheilosis) also in Vit.B2 and Iron deficiency, also caused by candida .
- **Cheilitis**: painful vertical fissures mainly of lower lip caused by malnutrition or with Crohn's disease.
- **Pallor, cyanosis** (See complexion)
- **Angioedema, herpes labialis.**

Teeth

- **Discoloration**: Tobacco, poor hygiene, lead poisoning
- **Loosing of teeth**: D.M.
- **Wide spacing**: Acromegaly.
- **Notched**: congenital syphilis (Hutchinson's teeth)
- **Dental caries, tooth extraction.**

Gum

- **Bleeding**: Vitamin C deficiency, thrombocytopenia, chronic liver disease.
- **Hypertrophy**: Epanutin, monocytic leukemia.
- **Blue line**: lead poisoning.

Tongue

The surface of the tongue normally varies as regard colour and appearance.

- A. **Color:**
- **Black**: iron therapy – Addison's.
 - **Brown**: smoking.
 - **Blue**: cyanosis.
 - **Pale**: anemia.
- B. **Atrophy**: (Glazed red tongue)
- With iron deficiency anaemia,
 - Hypovitaminosis e.g.: B12 \ pellagra.
- C. **Leucoplakia**:
- Due to chronic irritation, it is precancerous.
- D. **Moisture**:
- Dry tongue (under surface) = dehydration.
- E. **Strawberry tongue**:
- Scarlet fever
- F. **Macroglossia**

- Myxoedema
- Acromegaly
- Amyloidosis
- hemangioma
- Pseudomacroglossia:- Down
- Oedema :- Angioneurotic

G. **Neurological ex.**

- Tremors of the tongue
 - Thyrotoxicosis.
 - Parkinsonism.
 - Essential familial tremors.
- Percussion or tapping for:
 - Fasciculation.
 - Myotonia.
- Cranial nerve
 - For 5th \ 7th \ 9th \ 12th

H. **Scrotal or fissured tongue** Down syndrome and acromegaly.

J. **white coated** :- typhoid F

K. **Ptyalism** (increased salivation)

- Neurosis
- Stomatitis
- Reflex from GIT diseases.

Buccal mucosa, palate, tonsils and pharynx

1. Buccal mucosa

- Pigmentation (Addison's disease)
- Aphthus:- ulcers on the inner sides of the lips, the edge of the tongue and the inside of the cheek
- Koplik's spots

2. Tonsillitis and pharyngitis

3. Palate

- Jaundice appears early in the soft palate.
- petechial spots in thrombocytopenic purpura and leukemia.
- Pin point petechial spots also in infectious mononucleosis.
- High arched palate or cleft palate in congenital conditions.
- Palatal movement, palatal & pharyngeal reflexes (see neurology).

4. **Breath:**

- a. **Aceton** :- diabetic ketoacidosis
- b. **Ammonical** :- uremia
- c. **Foetor hepaticus** :- hepatic failure
- d. **Foiled smell** :- suppurative lung disease (Halitosis)
- e. **Other causes of halitosis** :- Bad oral hygiene, Sinusitis, Tonsillitis or Dyspepsia

Parotid enlargement

- Mumps
- Sarcoidosis
- Cirrhosis
- Endemic parotitis
- Stones or Tumor
- Hypoproteinaemia.
- Sjogren syndrome.

Neck

1. **General ex.**
 - a. Look for scars, lumps, rashes, hair loss, or other lesions.
 - b. Look for facial asymmetry, involuntary movements, or edema.
 - c. Palpate to identify any areas of tenderness or deformity.
2. **Pulsation** :- Arterial & venous. (See latter)
3. **Thyroid enlargement**:- (see latter)
4. **L.N.** :- (see latter)
5. **Torticollis** :- Hysterical, myositis of stemomastoid
6. **Rigidity** :- Meningeal irritation & cervical spondylosis
7. **Trachea** :- See the chest
8. **Supra sternal pulsations**:- (see latter)

9. **Neuromuscular examination of head and neck** :- see neurology

10. **Special Tests:**

- Facial Tenderness
- Sinus Transillumination
- Temporomandibular Joint.

Thrill over the carotid artery

Propagated from the heart	Not propagated from the heart
• AS • PS • PDA	• AR • A aneurysm

Causes of increased carotid pulsation = causes of suprasternal p

- A.R.
- Hyperdynamic circulation states e.g. Thyrotoxicosis
- Carotid aneurysm.
- After exercise or anxiety in thin persons.

Other Anatomical disorders

Congenital abnormalities ((e.g. high arched palate, webbing of neck & fingers & toes, pectus carinatum or pectus excavatum, arachnodactyly or polydactyly, pes cavus or flat foot, telangiectasia, neurofibromatosis))

1. **Marfan's syndrome:**

H&N	• Ectopic lens ± cataract • High arched palate.
Extremities	• Thin and Long finger (arachnodactyly) • encircling test • Flat foot or pes cavus.
Chest	• Pectus excavatum or pectus carinatum • Kyphoscoliosis. • Associated Ht. Lesions: • Dissecting Aneurysm of aorta \ A.R. \ A coarctation • Mitral valve prolapse M.I., • AV bundle conduction abnormalities.
Skeletal	• Loose subluxated Joints. • High stature.

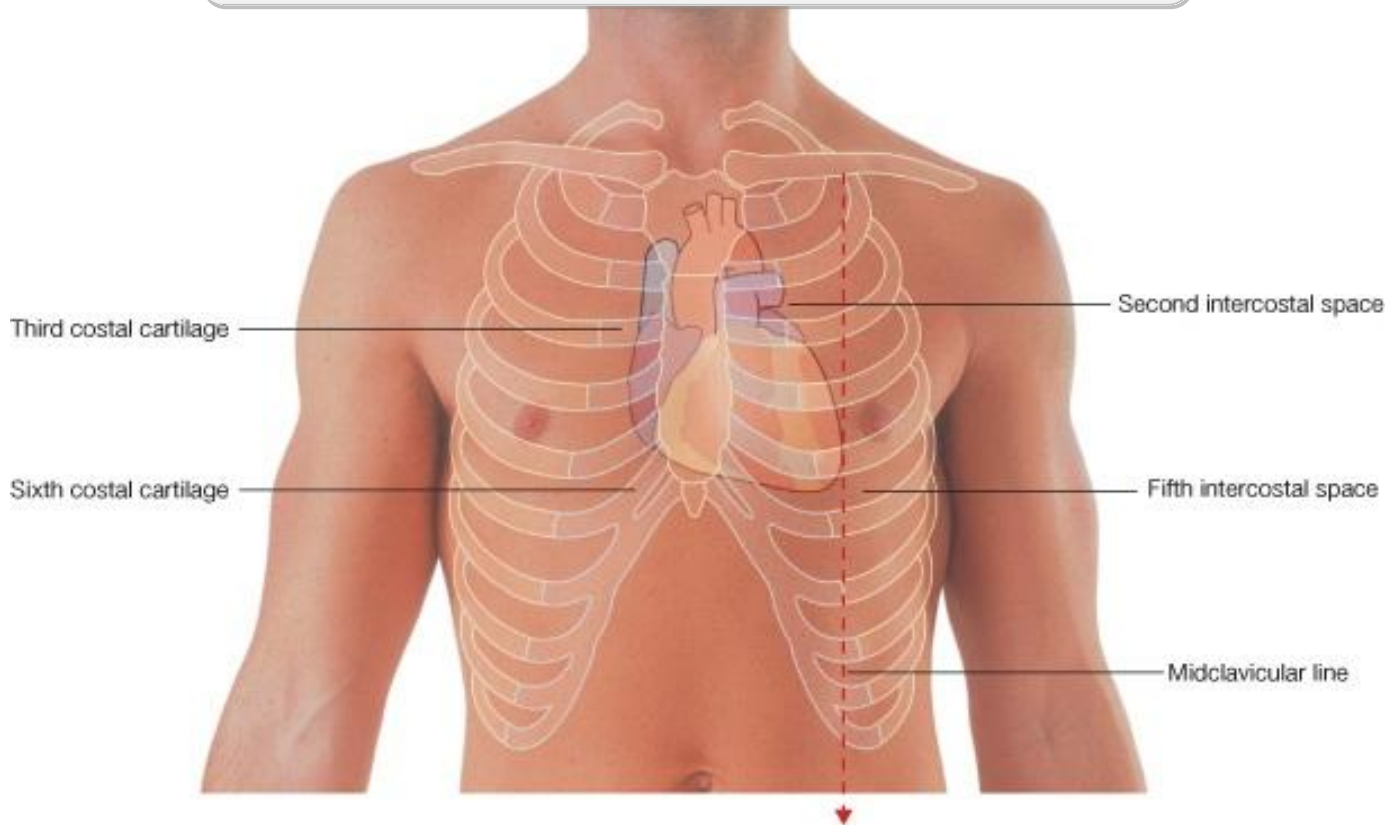
2. **Hereditary telangiectasia** : with pulmonary A-V. fistula

3. **Neurofibromatosis** : with pheochromocytoma
4. **Polydactyl** : with ASD
5. **Low set ear & depressed nose** : in cong. A.S.
6. **Some Genetic defects** : Down's syndrome: (A.S.D.) -Turner's syndrome: (coarctation of aorta).

CARDIOLOGY



SURFACE ANATOMY OF THE HEART



- The upper limit of the heart reaches as high as the third costal cartilage on the right side of the sternum and the second intercostal space on the left side of the sternum.
- The right margin of the heart extends from the right third costal cartilage to near the right sixth costal cartilage.
- The left margin of the heart descends laterally from the second intercostal space to the apex located near the midclavicular line in the fifth intercostal space.
- The lower margin of the heart extends from the sternal end of the right sixth costal cartilage to the apex in the fifth intercostal space near the midclavicular line.

Surface anatomy of the cardiac valves

- P (pulmonary): deep to the left 2nd sterno-costal junction.
- A (Aortic): opposite the left 3rd intercostal space.
- M (mitral): deep to the left 4th sterno-costal junction. So the above three valves present behind the left border of the sternum.
- T (tricuspid): behind the center of the sternum opposite the left 5th intercostal space.

To listen for valve sounds, position the stethoscope downstream from the flow of blood through the valves

- The tricuspid valve is heard just to the left of the lower part of the sternum near the fifth intercostal space.
- The mitral valve is heard over the apex of the heart in the left fifth intercostal space at the midclavicular line.
- The pulmonary valve is heard over the medial end of the left second intercostal space.
- The aortic valve is heard over the medial end of the right second intercostal space.

HOW TO DEAL WITH A CARDIAC

We deal with cardiac case easily, we take history, do examination, ask for investigations and treatment then we give our provisional diagnosis.

History

As before (don't forget)

1. Personal History
2. Complaint
3. History of present illness
4. Past history
5. Family history
6. Socioeconomically state
7. In females (Menstrual and obstetric history)

Examination

As before (don't forget)

- General examination as before but focus on the things related to cardiology "or leave it for local examination"

Overview	
A أرقام	1. Pulse (<i>see before in details</i>)
	2. Temperature
	3. Blood pressure (<i>see before in details</i>)
	4. Respiratory rate
B	5. B uilt
C	6. C olor
D	7. D ecubitus
E حاجة فوق وحاجة تحت وحاجة في النص	8. Neck vein – H & N (total) (<i>see before in details</i>)
	9. Clubbing (upper limb)
	10. Lower limb edema (lower limb)
F	11. Mentality "فكر"
	12. Face + general look

- Local examination (cardiac examination)

Investigations

Don't forget these 4 items:

1. X-ray:

- Chamber enlargement.
- Pulmonary congestion in left sided diseases.
- Pleural effusion.

2. ECG:

- Chamber enlargement.
- Detect the cause.

3. Echocardiography:

- Chamber enlargement.
- Detect the cause.
- Paradoxical movement of the myocardium.

4. Catherterization:

- Chamber enlargement.
- Detect the cause.

N.B.

We add some investigations in certain cases like blood culture in case of infective endocarditis...etc.

Treatment

1. Treatment of the cause.
2. Treatment of the precipitating factors.
3. Specific treatment.

N.B.

Don't forget in case of valvular lesion:

- Medical treatment.
- Surgical treatment.
- Some times (balloon dilatation).

So, let's start with the cardiology branch 😊

SYMPTOMS OF CARDIOLOGY

Symptoms of the cardiology are

- 4C.
- 4P.
- Others.

C	P	Others
1. Pulmonary Congestion. 2. Systemic venous Congestion. 3. Low Cardiac output. 4. Cyanosis.	1. Palpitation. 2. Pain. 3. Pressure manifestations. 4. Blood pressure changes.	1. Fever. 2. Thrombo-embolic manifestations.

Natural history

1. P congestion found to be the 1st C\O in MS & the last C\O in other left sided valve lesions.
 2. Aortic valve lesion usually presented with Chest pain??
 3. Regular palpitation = regurge
- **MS** = P. Congestion → P.H. (Low COP) → Rt.V.F (S. Congestion) + A.F. (irregular Palpitation)
 - **MR** = Palpitation → Lt.V.F. (P. Congestion)
 - **AS** = Low COP → Lt.V.F. (P. Congestion)
 - **AR** = Palpitation → Lt.V.F. (P. Congestion)
 - **TR** = Palpitation → Rt.V.F (S. Congestion) on top of Mitral lesion.

N.B.

- ✓ **Double mitral:** - Regular Palpitation on top of P. Congestion or reverse
- ✓ **Irregular palpitation** ---- AF ---- MS or Double M

Pulmonary congestion

Caused by: - M.S. \ LVF

Including:

(dyspnea, cough, haemoptysis & recurrent chest infection)

Dyspnea

Is abnormally uncomfortable awareness of the act of breathing.

1. Exertional

2. Postural

- **Orthopnea** -> dyspnea on lying flat relieved with erect position.
- **Platypnea** -> dyspnea on erect position
- **Trepopnea** -> dyspnea on lying on one side.

3. Nocturnal

Pathogenesis of cardiac dyspnea

A. Mechanical factors:

- Pulmonary congestion:** (leading to ↓ lung compliance) occurs in left sided heart failure or lesion leading to:
 - Decreased lung compliance due to interstitial edema (the most important factor).
 - Diminished alveolar capacity with transudation in some alveoli.
 - Congestion of bronchial mucosa with or without bronchospasm.
- Infra-diaphragmatic causes:**
Pericardial effusion, constrictive pericarditis and right sided failure with SVC lead to enlarged liver and ascites which decrease diaphragmatic mobility.
- Fatigue of respiratory muscles:**
Decreased respiratory muscles' perfusion due to low cardiac output.
- Pericardial effusion and pleural effusion (with heart failure):**
Leading to mechanical compression of the lungs.

B. Nervous factors:

a. Activation of Hering – Breuer reflex:

This is a normally present reflex in which impulses arise from stretch receptors present in the terminal air passages at the end of inspiration, this leads to reflex inhibition of inspiratory center and passive relaxation of the chest → expiration.

In left sided failure, interstitial oedema activates this reflex, causing shallow rapid breathing.

b. Churchill – cope reflex:

It occurs in pulmonary venous congestion which leads to reflex stimulation of respiratory center, through the juxtacapillary receptors of the lung which are stimulated due to pulmonary venous congestion (high pulmonary capillary pressure).

C. Chemical factors:

Pulmonary venous congestion and diminished tissue perfusion (↓ COP) lead to hypoxia, which stimulate respiration.

Complaint: -

Shortness of breath (SOB) – exertional

السؤال / هل عندك نهجان أو كَرشَة نفس؟؟

NYHA classification of dyspnea

I	Dyspnea on more than ordinary effort
II	Dyspnea on ordinary effort
III	Dyspnea on sub ordinary effort
IV	Dyspnea on Rest

- **Orthopnea:** - Dyspnea on lying flat & relieved on erect position.

تسأل المريض بينام على كام مخدة؟؟

Paroxysmal nocturnal dyspnea (PND)

Dyspnea, cough ± wheeze developed **1-2 hours** after sleep

Spontaneously resolved called the Cardiac Asthma

بتسأل ،، لما بتنام بتكمل نوم للصبح ولا بتصحى؟؟

ولو بتصحى ،، إمتى؟؟ وعلى إيه؟؟

Mechanism of PND

1. Increased V.R. during sleep leading to aggravation of pulmonary congestion.
2. Absorption of oedema fluid into the circulation causing further increase in V.R.
3. Diminished Sympathetic activity during sleep causing reduction of cardiac contractility.
4. Night mares lead to tachycardia and elevation of BP.
5. Slipping down from high pillows.

N.B.

- PND is highly specific for cardiac cases.
- Diagnostic for left sided HTF.
- But we have to exclude B.A.

	Cardiac A.	Bronchial A.
Age	any age	young age
Other symptoms	+ cardiac symptoms	chest symptoms
Duration	short duration	long
Time of attack	1 - 2 hrs after sleep	Early in the morning
Relieved	Spontaneously	Bronchodilators
Dyspnea	Inspiratory	Expiratory
Sputum	Frothy (may be blood tinged)	Thick.

Systemic congestion

In right ventricular failure (M.S. / T.R.)

Manifested by:-

1. **Oedema L.L. usually before ascites**

LL swellings

Ascites precox = ascites before LL oedema in cases of pericardial & tricuspid diseases.

2. **Hepatic congestion:**

Pain in right hypochondrium + Jaundice.

3. **G.I.T congestion** = Dyspepsia.

Q : Jaundice in cardiac patient?

1. Associated viral infection.
2. Haemolytic Jaundice = pulmonary infarction or metallic valve
3. Hepatocellular Jaundice = hepatic congestion (cardiac cirrhosis)
4. Obstructed Jaundice = hepatic congestion obstruct the biliary tract

Low cardiac output

فوق	Syncope Headache. Blurring of vision + Syncope. Dizziness
متتصف	Anginal pain Kidney = Oliguria
تحت	Muscle = easy fatigability Skin = pallor /cold.

The key word is **Syncope**

Syncope: sudden transient complete loss of consciousness due to reduced cerebral blood flow. If the ischemia prolongs, convulsions may occur. It is associated with postural collapse with spontaneous recovery.

Complaint:

Fainting or Black out.

Etiology of syncope

1. Vasomotor syncope.

A. Vasovagal syncope (neurogenic syncope):

- It results from severe vagal stimulation which leads to: *severe bradycardia, hypotension, pallor & sweating.*
- It results from: *sudden severe fear, pain, and trauma (e.g. to testicles).*
- It is the most common cause of syncope & is known as simple fainting.

B. Carotid sinus syndrome:

- It results from *pressure on hypersensitive carotid sinus baroreceptors: e.g. during shaving.*

2. Cardiac syncope

(Any cause of low COP) especially:

- **A**ortic stenosis (or any other valvular obstruction).
- **A**cute heart failure (e.g. AMI).
- **A**rrhythmias (whether tachy- or brady-arrhythmia).
- **A**dams-stokes attacks.

3. Cerebral syncope

(Reduced cerebral blood flow)

- Vertebrobasilar TIAs.

4. Hypoxic syncope

(↓ O₂ content of the cerebral blood flow)

- Fallot's tetralogy & other cyanotic diseases or severe anemia.

5. Postural syncope

(Orthostatic syncope)

- Normally, reflex VC of blood vessels of L.Ls occurs on standing to prevent pooling of blood in lower limbs.
- This effect is mediated through sympathetic stimulation.
- If this mechanism is defective, BP will be markedly lowered in the standing position (postural hypotension) & syncope may occur.

Causes include:

- Autonomic neuropathy, **e.g.** Diabetes.
- Sympatholytic drugs, **e.g.** ganglion blockers & vasodilators.
- Lumbar sympathectomy.
- Hyponatremia.
- Prolonged recumbency.
- Elderly patient.
- Huge varicose veins.
- Hypovolaemia. **e.g.:** Haemorrhage or dehydration.
- Weakness of the muscles of the lower limbs (muscle pump).

6. Situational syncope

- Rare syncope caused by a variety of activities in susceptible individuals:
 - ✓ Cough syncope (*Tussive syncope*).
 - ✓ Micturition syncope (*more common in old men especially at night*).
 - ✓ Defecation syncope.
- Underlying mechanism:
Straining → decreased VR → decreased COP → syncope.

N.B.

- *Cardiac syncope*
 1. Usually exertional
 2. Not accompanied by convulsions

Cyanosis

- a. **Age of onset:**
 - Since birth = Fallot's tetralogy.
 - Few years after birth = Fallot's triology.
 - In teenager = Eisenminger's syndrome (reversed shunt).
 - Above age of 40 years = COPD with or without Corpulmonale.
- b. **Cyanotic spells and squatting** = Fallot's tetralogy.
- c. **Differential cyanosis** = P.D.A with reversed shunt.
- d. **Exertional cyanosis** = cases of cardiac shunts / cases of a cyanotic Fallot's / interstitial pulmonary fibrosis.

Pain

Don't forget 11 points

1. **Onset:** sudden for example; anginal pain
2. **Course:** intermittent.
3. **Duration:** 30 sec. → 30 min.
4. **Association:**
 - Angor animi.
 - Dyspnea.
 - Sweating.
5. **What increase ↑ ↑ :**
 - Exercise.
 - Sexual intercourse.
 - 5 Hs.
 - ✓ **H**heavy meal.
 - ✓ **H**heavy smoking.
 - ✓ **H**ypothermia.
 - ✓ **H**igh attitude.
 - ✓ Stress **م** **ال**
6. **What decrease ↓ ↓ :**
 - Rest.
 - Sublingual nitrate.
7. **Effect of treatment:** Respond

8. **Last attack**: Describe it.
9. **Site**:
 - Retrosternal.
10. **Radiation**:
 - Left shoulder.
 - Jaw.
 - Back.
 - Epigastric.
11. **Character**: any type except stitching.

N.B.

Anginal pain never to be:

- *Localized.*
- *Infra mammary.*
- *Stitching.*
- *> 30 min.*

Pressure manifestation

The most posterior chamber of the heart is the **Lt Atrium**, which is markedly enlarged in M.S. /M.R. and press on

Pressure symptom

- **On trachea** = brassy cough.
- **Bronchial** = dyspnea
- **Oesophagus** = dysphagia
- **Left recurrent laryngeal nerve** = hoarseness of voice (Ortner's).
- **SVC** = oedema and cyanosis of face and U.L.

Palpitation

Complaint

Awareness of heart beats

Cause

1. Rapid heart rate. e.g.: Sinus or paroxysmal tachycardia.
2. Forcible heart contraction (volume overload). e.g.: A.I. or M.I.
3. Irregular heart, e.g.: extrasystole or A.F.
4. Cardiac neurosis.

Ask about

- Is it regular or irregular?
- Precipitating factors.

Manifestations of hypertension

Hypertension symptoms:

- Asymptomatic
- Headache.
- Blurring of vision.
- Tinnitus.
- Epistaxis.

N.B.

No symptoms of diagnosis of hypertension, only history of regular use of anti hypertensive drug.

Embolic manifestations

Embolus

Insoluble material in circulation

origin

Atrium	Valve	Ventricle	Vessel
Left atrium in M.S. and A.F.	Vegetation in infective endocarditis	Myocardial infarction	Aortic atheromatus plaque

Effect:

Cerebrum	Coronary artery	Peripheral artery	Eye	Renal artery	Superior mesenteric artery
Hemiplegia.	Chest pain.	• Pulsless. • Pallor. • parasthesia or • Paralysis.	Blindness	Painless haematuria.	Abdominal pain (intestinal obstruction)

Fever in cardiology

Aetiology

Cardiac	Vessels	Lung
a. Endocardium • Infective endocarditis (serious). • Rheumatic fever (commonest). b. Myocardium • Myocarditis. • Myocardial infarction. c. Pericardium • Pericarditis. • Effusion.	• Thrombophlebitis. • D.V.T.	• Pulmonary embolism. • Pulmonary infection.

Drugs: 6 Cs

- Corticosteroids.
- Licorice.
- Contraceptives.
- Amphetamine and thyroxine.
- Carbenoxolone.
- Anti Common Cold drugs.
- Cyclosporin.

Drugs that cause Hypertension

Symptoms of the other systems

SUMMARY OF HISTORY OF PRESENT ILLNESS IN C.V.S

Pulmonary congestion

- هل عندك نهجان (كَرْشَة نفَس) ؟؟
- بتنام على كام مخدة ؟؟
- بتكمل نوم للصبح ولا لا ؟؟؟
- فيه كُحَة أو إتهابات على الصدر متكررة ؟؟؟
- هل البلغم معرق بدم ؟؟؟

Systemic venous congestion

- رجلك أو بطنك نفخت ؟؟؟
- فيه ألم في جنبك اليمين ؟؟؟

↓ *Cardiac output*

- هل عندك صداع أو دغلة أو إغماءات ؟؟؟
- لما تمشي فيه وجع في عضلاتك ؟؟؟

Cyanosis

- إزرقيت ؟؟؟ (حصل زُرقة) ؟؟؟

Pain

- عندك ألم في الصدر ؟؟؟

Pressure manifestations

- هل صوتك اتبع ؟؟؟
- عندك صعوبة في البلع ؟؟؟

Palpitation

- بتحس بضربات قلبك ؟؟ (رُفرة) ؟؟؟

Blood pressure changes

- بتأخذ أدوية للضغط ؟؟؟

Embolic manifestations

- حصل ثقل في لسانك ؟؟؟ أو إيدك ؟؟ أو رجلك ؟؟

Fever

- حصل إرتفاع في درجة الحرارة ؟؟؟

وتسأل المريض برودو

وتقول له :

هل عندك مشاكل في أي جزء ثاني في جسمك ؟؟؟

عملت فحوصات إيه ؟؟

وأخذت علاج إيه ؟؟؟

PAST HISTORY

Diseases: -

Rheumatic fever.

EXAMINATION OF OTHER SYSTEMS IN CARDIAC CASE

Chest

Cause	Result	Association
Corpulmonale	1. Chest infection. 2. Pleural effusion. 3. Crepitations.	Kartagner's syndrome = Bronchiectasis + dextrocardia + absent frontal air sinus

Abdomen

Spleen

1. **Palpable** = cirrhosis.
2. **Palpable & tender** = infective endocarditis.

Ascites

- Normal sequels
- Ascites precox = pericardial disease - T.R.

Liver

1. **Palpable** = Liver cirrhosis (Cardiac cirrhosis - B fibrosis)
 2. **Palpable + tender** = congested liver = Rt HF / Pericardial diseases.
 3. **Palpable + tender + pulsating** = pulsating liver = T.R. (systolic) / T.S. (diastolic).
- ☺ Other causes of pulsating liver

Highly vascular hepatic tumor or from aortic aneurysm.

VALUE OF GENERAL EXAMINATION IN C.V.S.

A. رقام

- Pulse.
- Blood pressure.
- Temperature.

B. Built

- Under built
 - ✓ In congenital heart disease. Or
 - ✓ Rheumatic disease since childhood.
- Over built
 - ✓ Generalized edema.

C. Colors

- Pallor
 - ✓ Associated anemia.
 - ✓ Edema (appears pale).
 - ✓ Shock.
 - ✓ ↓ C.O.P.
 - ✓ Rheumatic fever.
 - ✓ Infective endocarditis.
- Cyanosis
 - ✓ Central
 - Congenital heart disease.
 - Hypoxic.
 - Cor pulmonale.
 - ✓ Peripheral
 - Systemic venous congestion.
- Jaundice → 4 causes???

D. Decubitus

- Orthopnea.
- Squatting.
- Praying position.

E. حاجة فوق وحاجة تحت وحاجة في النص

- neck vein -- فوق

Clinical significance of neck veins

1. Normally:

They are non congested, & are pulsating with systolic collapse.

2. Abnormally:

a. **Abnormal pressure** "congested neck vein"❖ Pulsating:

- ✓ Right sided heart failure.
- ✓ Pericardial effusion & constrictive pericarditis.
- ✓ Hypervolemia.

❖ Non-pulsating:

- ✓ SVC obstruction (SVC thrombosis or Mediastinal syndrome).

b. **Abnormal pulsations** "abnormal waves"❖ a-wave:

- ✓ Absent: AF.
- ✓ Giant: pulmonary hypertension, PS, TS.
- ✓ Cannon waves:

- ✚ Regular: nodal rhythm.

- ✚ Occasional: A-V dissociation.

❖ Giant V:

- ✓ obliterated (systolic expansion of neck veins):

- 1) TR.

- 2) AF.

- 3) Constrictive pericarditis.

- Pericardial effusion.

- 4) Rt. H.f.

• **النص clubbing**

- ✓ Pale

- ✚ Infective endocarditis.

- ✚ Left atrial myxoma.

- ✓ Blue

- ✚ Congenital heart disease.

• **تحت lower limbs**

- ✓ Right sided heart failure.

- ✓ Pericardial disease.

DIAGNOSIS

The diagnosis should include the following four categories:

1. Aetiological

- **Rheumatic:** multivalvular – history of rheumatic fever.
- **Congenital:** since birth + anomalies.
- **Ischemic.**
- **Hypertensive.**
- **Surgical.**

2. Anatomical

- **Valve lesion.**
- **Pericardium:** constrictive pericarditis.
- **Myocardium:** cardiomyopathy.

3. Pathological

- Stenosis. Or
- Regurge. Or
- Double.

4. Functional

- **Compensated:** no manifestations of LVF or RVF.
- **Non-compensated:** manifestations of LVF or RVF.
- Or **complicated by:**
 - ✓ A.F. or any arrhythmia.
 - ✓ Embolic manifestations e.g. hemiplegia.
 - ✓ Infective endocarditis.
 - ✓ Chest infection.

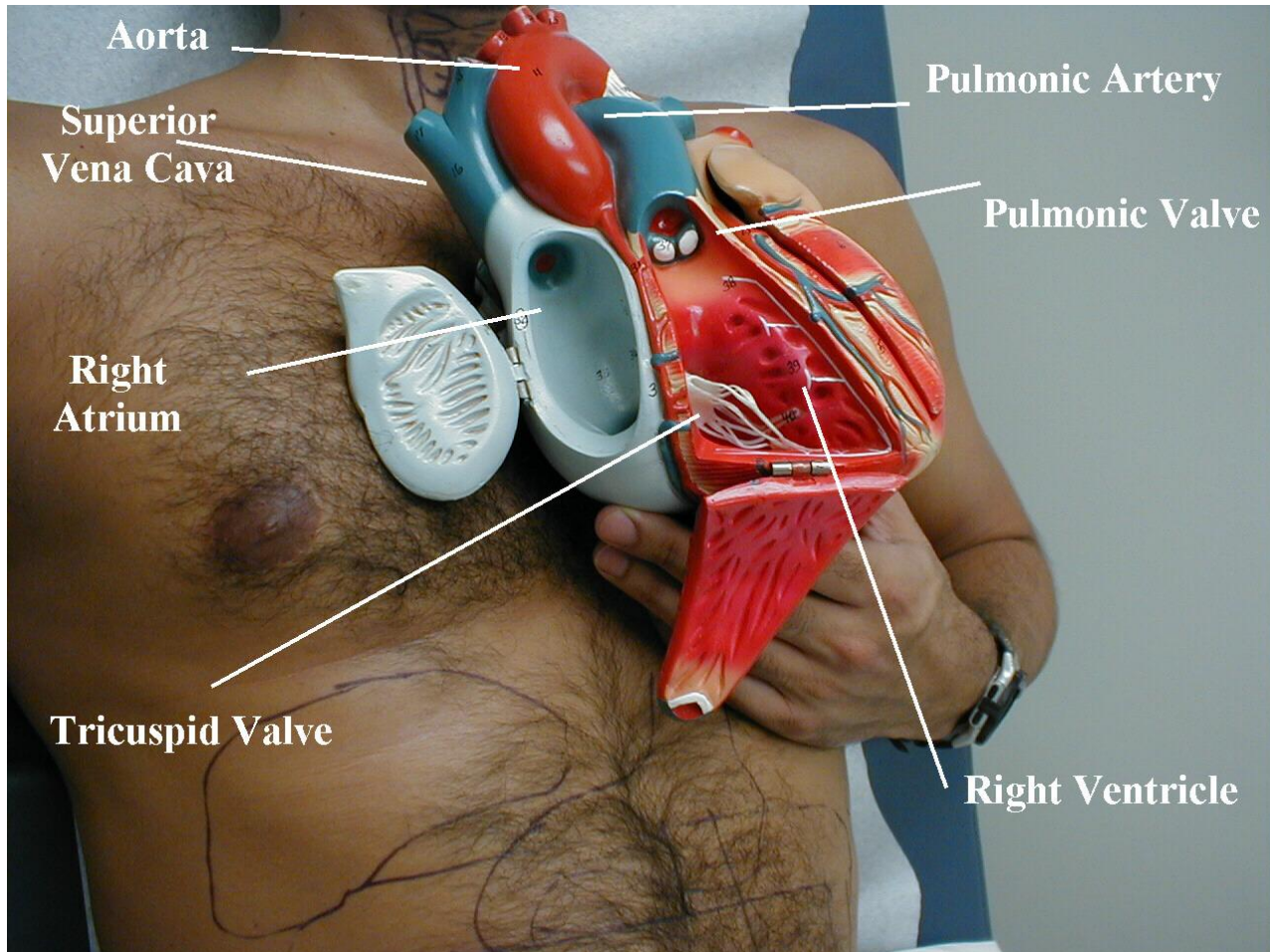
Examples

- *Rheumatic heart disease (M.S. – M.I.) compensated, non-complicated.*
- *Rh. Heart disease (M.I. - A.I.) left sided heart failure complicated with infective endocarditis.*
- *Congenital Heart disease (V.S.D.) compensated with no complications.*

LOCAL EXAMINATION

Don't forget these 4 items

1. *Inspection.*
2. *Palpation.*
3. *Percussion.*
4. *Auscultation.*



PATIENT POSITIONING:

- Expose the patient's chest up to the umbilicus.
- Position the patient supine with the head of the table slightly elevated.
- Always examine from the patient's right side.
- Make sure that the patient is comfortable in this position.

Value of Precordial examination:-

1. Evidence of right ventricular enlargement:
 - a. Precordial bulge
 - b. apex beat: (outermost lower most impulse)
 - Diffuse.
 - Shifted outwards.
 - Shows systolic retraction: accompanied by reciprocal left parasternal uplift (**right ventricular rocking**)
 - c. Left parasternal & epigastric pulsation.
 - d. Dullness over the lower half of the sternum.
2. Evidence of left ventricular enlargement:
 - a. Apex beat:
 - Localized.
 - Shifted outward & downward.
 - Shows systolic bulge: accompanied by reciprocal left parasternal retraction (**left ventricular rocking**)
 - b. Apex impulse:
 - Heaving (forcible, sustained): pressure overload.
 - Hyperdynamic (forcible, non-sustained): volume overload.
 - Hypodynamic (weak): myocardial disease.
3. Evidence of great vessel dilatation:
 - a. Dilated pulmonary artery:
 - Dullness & pulsation in the second left space.
 - b. Dilated aorta:
 - Dullness & pulsation in the second right space.
4. Evidence of endocardial affection (valvular affection):
 - Presence of specific murmurs & thrills.
5. Evidence of myocardial affection:
 - Presence of S₃ gallop over the mitral or tricuspid areas in LV or RV affection respectively.
6. Evidence of pericardial affection.
7. Evidence of pulmonary hypertension:
 - a. Precordial examination:
 - Pulsations, diastolic shock & dullness in the second left space.

b. Auscultation:

- Accentuated pulmonary component of the second heart sound.
- Ejection systolic click.
- Ejection systolic murmur due to relative pulmonary stenosis.
- Early diastolic murmur due to functional pulmonary regurge (Graham Steel).
- S₄ over the tricuspid area.

8. Evidence of arrhythmias:

- Change in the rate or rhythm of the heart beats.

Inspection

Observe the chest carefully through tangential view.

Specifically note for:

- Shape of chest & chest wall lesion :- (See chest)
- Dilated vein on chest wall > (S.V.C. obstruction).
- Scar of previous operation
 - i. **Median sternotomy** (open heart surgery) **e.g.**; valve replacement or coronary bypass.
 - ii. **Inframammary = lateral thoracotomy** (closed heart surgery) **e.g.**: mitral valvotomy.
- Precordial bulge:- indicates cardiac enlargement during childhood **e.g.** R.V. enlargement - pericardial effusion.
- Pulsation of different area (mainly for apical pulsation).

Palpation

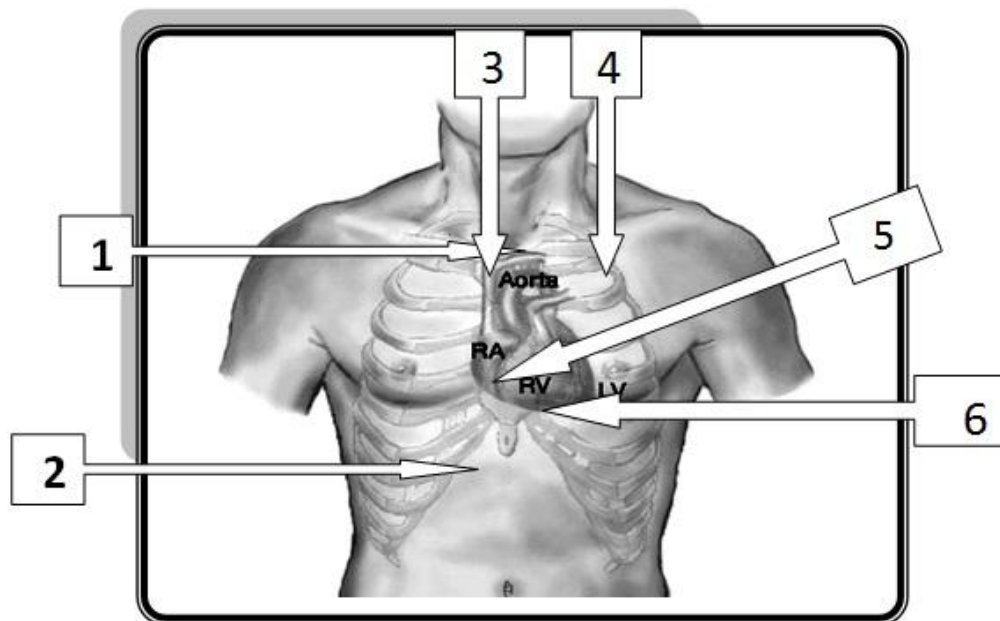
Usually inspect and palpate at the same time

Palpation for detection of:-

1. Pulsation
2. Thrill
3. Palpable sound

*pulsation***Areas of palpation**

Area	Aetiology	Technique
1- Supra sternal	A.R. "Corrigan sign" + thin person + Hyperdynamic state Aortic aneurysm	Place your index / semi setting pt.
2- Epigastric P. :- Place your hand longitudinal in the subcostal angle Pulsation classified according to the direction into		
A) at tip of fingers	RV++	Increased with deep inspiration
B) from the right side	Hepatic P (TS / TR)	Enlarged & Tender during bimanual ex.
C) from behind	Aortic P (thin/ Hyperdynamic/aneurysm)	Pulsating down to umbilicus
3- Aortic area	Hypertension A. aneurysm / S	By tip of fingers (TV)
4- Pulmonary area	PH++ / P. anysm.	
5- Rt. parasternal	Rt. Atrium++ / Lt. at.	By tip of fingers (Vert) \or palm of hand
6-Lt. parasternal	R.V. ++ / Lt. atrium	By base of hand (increased with expiration)



7) The apex.

Technique

(Inspection – palpation – left lateral position)

- Firstly, by inspection
- Place your hand over the left hemi-thorax region
- Feel for the outer most and lower most pulsation.
- Left lateral position (for detection of weak P)
- Count the intercostal spaces (first identify the angle of Louis = the rib attached alongside this is the 2nd rib and the space below the rib is the 2nd space).



N.B.

Percussion or auscultation may be used for detection of absent apex.

Apex

Produced by the anterior movement of the left ventricle during early systole, occurs during isometric contraction of the left ventricle.

The normal apex felt as a **GENTLE NONSUSTAINED TAP**

N.B.

the point of maximal impulse may not be the apex as in rheumatic mitral valve stenosis the PMI may be the right ventricle.

Apex = Outermost and lowermost visible and palpable part of the heart.

تمثيلية ال apex

النظر إلى ال apex مماسياً ، ثم وضع اليد كاملة على ال apex
ثم محاولة تحديدها بمجموعة من الأصابع ، ثم بإصبع واحد
وتحدد مكانها ، ثم زحزحة اليد اليسار قليلاً
وأقلب المريض يساراً وأحدد صفتها
" نظرة – لمسة – عدة – لفة "

Question ☺

Absent apex: (OPERA):-

Definition: Not visible or palpable apex even on left lateral position.

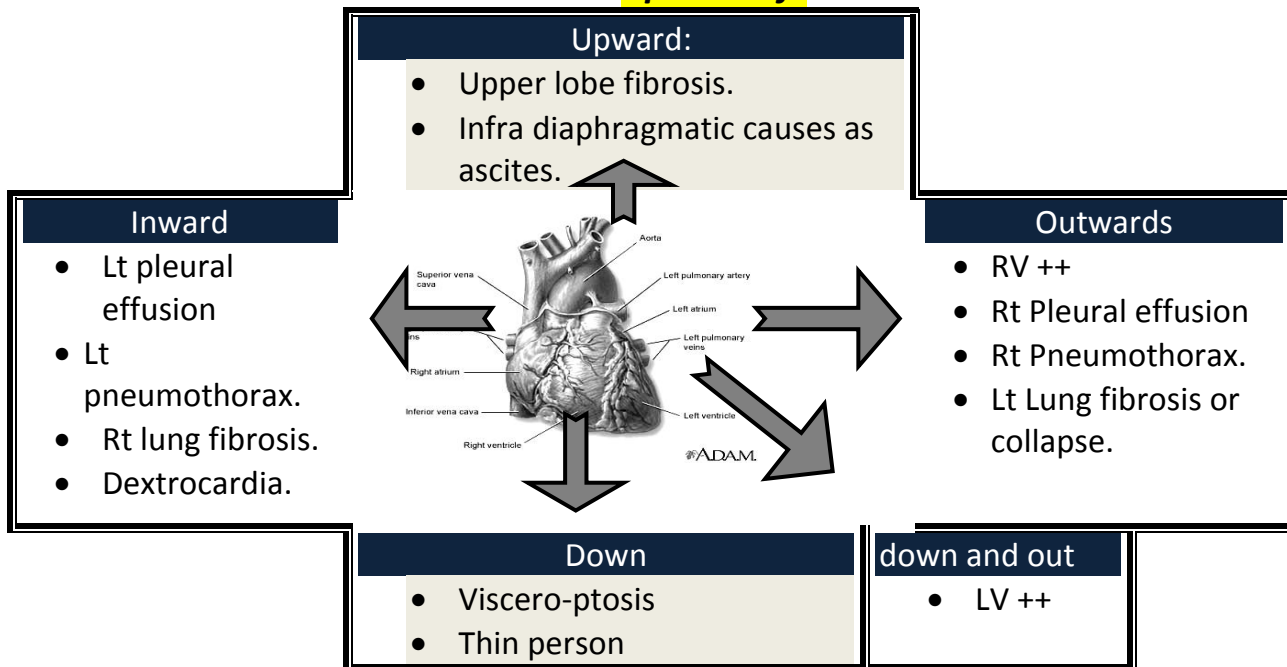
- O → **O**besity.
P → **P**ericardial effusion – pleural effusion.
E → **E**mpysema.
R → **R**ib "under rib".
A → **A**nomalies "dextrocardia".

Comment on apex

1. Site :- 5th / Lt. MCL
2. Area.
3. Ch.ch.
4. Thrill
5. Rate "P. deficit".
6. Rhythm.
7. Rocking.

(1) Site

Normally in the Lt. **5th** intercostal space just **inside the M.C.L** (3.5 inch from the midline)

Apical shift**(2) Area "extend"**

Normally it is localized (less than **one inch** / occupies **one space**).

Diffuse apex: - Right ventricular enlargement

The apex diameter is more than one inch or more than one space or the apex with ill-defined medial border

Localized apex: - Left ventricular pulsation

The apex diameter is less than one inch or in one space or the apex with well defined medial border.

(3) Character

Defined as: -

Force and duration of the apex

Hyperdynamic = forcible (In left lateral)

Volume overload as A.R. / MR / VSD.

Heaving = Sustained.

Tension overload as systemic Hypertension / AS / A coarctation

Slapping = papable S1 MS.

(4) Thrill & palpable sounds

Thrill

- Diastolic thrill in M.S.
- Systolic thrill in M.I.

Palpable sounds 1st H.S.:- in M.S.

(5) & (6) Rate & Rhythm

Normal: - Regular with HR equal to the radial pulse.

Irregular = (AF/ extra systole) / Apical rate for counting of P. deficit.

(7) Rocking

Left ventricle ++:- Apical bulge + left parasternal retraction (**anti-clockwise**)

Right ventricle ++:- the reverse (**clock-wise**).

Double apex:

- Ventricular aneurysm:** - systolic – diastolic pulsation (paradoxical).
- Hypertrophic Cardiomyopathy:** - systolic – systolic pulsation.

Thrill

Better to be detected by the palm

Apex	Left para sternal	Base	
- Systolic = MR - Diastolic = MS	Systolic = VSD	Aortic area - Systolic = P.S. - Continuous = PDA	Pulmonary area - Systolic = P.S. - Continuous = PDA

Palpated sound

Better to be detected by the tip

1. Apex
 - Palpable S_1 in **M.S.**
 - Palpable S_3 or S_4 .
 - Palpable rub.
2. Aortic area
 - Palpable A_2 "syphilitic **A.R.**".
3. Pulmonary area
 - Palpable pulmonary component of S_2 in PH^{++} .

Percussion

Of no value in the recent medicine

1. **Hepatic dullness**
 - Start from the 2nd space downward on the Rt MCL by heavy percussion
 - When you reach the hepatic dullness ask the pt. to breath in (Tidal percussion)
 - Upper border of the liver normally in the 4th or may be in the 5th space.
2. **Right border**
 - Precede one space above i.e. 3rd space or 4th
 - Percuss parallel to the right border of the sternum (from out to inward)
 - Normally no dullness to the right of the sternum
 - Dullness denotes:- RA^{++} , A aneurysm or pericardial effusion.
3. **Aortic a**
 - Percuss the 2nd Rt space from lateral to medial (from MCL to the sternum).
 - Normally it is resonant up to the sternum

Dullness means:- A aneurysm (pulsating) Post stenotic dilatation (No pulsation)

4. **Pulmonary a**
 - Percuss the 2nd Lt Space from lateral to medial (from MCL to the sternum).
 - Normally 1.5 cm of dullness may be detected

Increased dullness means:- PH^{++} (pulsating) P. dilatation (No pulsation)

5. **Waist** (on request)
 - Percuss the 3rd left space
 - Normally there is a1 finger of dullness.

Increased dullness (obliterated waist):- LA^{++} .

6. Outside the apex

- Percuss the apex from outer to inner at the same space
- Normal:- No dullness outside apex

Dullness outside the apex: - Pericardial effusion

7. Retrosternal

- Percuss the lower 1/3 of the sternum directly by light percussion
- Normally impaired note

Dullness: - R V ++

8. Bare area of the heart:

Def. Area of heart not covered by lung

"**Normally dull**" (see the chest)

Shifting dullness:

In pericardial effusion

Over the pulmonary area = Dullness at flat position disappear with sitting.

Auscultation

Technique

1. Stethoscope.
2. Site of auscultation.
3. The maneuver of auscultation.

Auscultatory findings

1. Heart sounds: S1 & S2
 2. Additional sounds: - S3, S4, opening snap and clicks.
 3. Murmurs.
- ± Pericardial rubs / L. crepitations.

a) **Ideal stethoscope**

1. Optimal stethoscope tubing length is twelve inches (30 cm)
2. Make sure that the earpieces are fit in the external ear.
3. Make sure no air leaks occur between the chest wall and the stethoscope earpiece.

The Cone (bell)

is best listened to low pitched sounds e.g.: S3 / S4 / rumbling murmur of MS.

The diaphragm

Identifies high pitched sounds e.g.: normal heart sounds and the murmur of aortic incompetence.

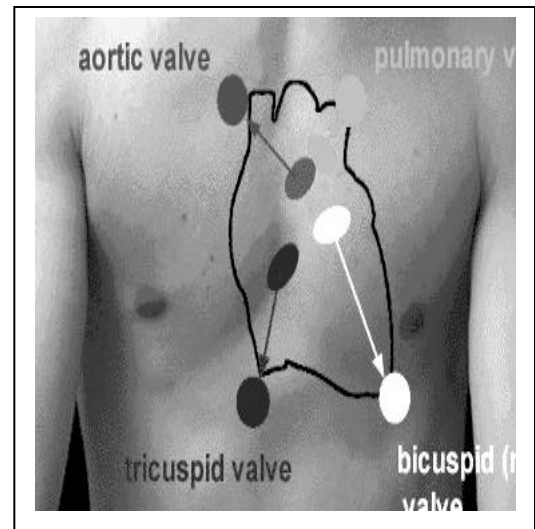
Remember

The anatomical sites of valves

- **P** = 2nd left sternal border.
- **A** = 3rd left sternal border.
- **M** = 4th left sternal border.
- **T** = 5th left sternal border.

N.B.

Pulmonary & tricuspid valves are anterior
So, heard at their anatomical sites
But the **aortic & mitral** valves are posterior
So, heard at the direction of blood flow.

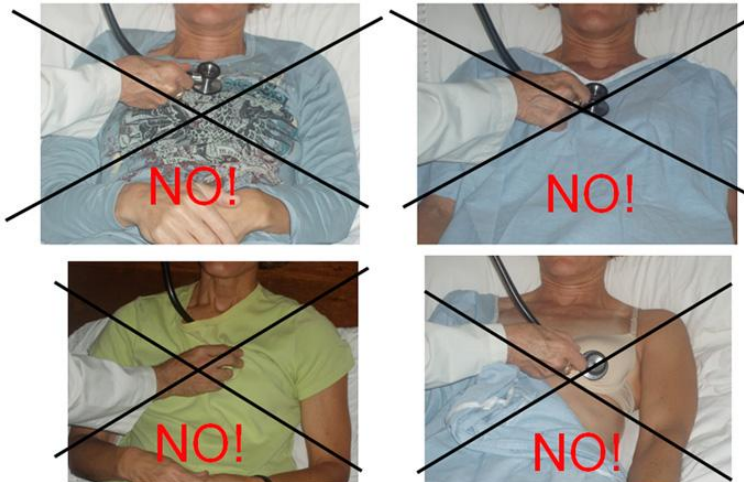


b) Site of auscultation

- **Mitral:** - apex (lt 5th space at the MCL)
- **Tricuspid:** - at lower end of the sternum
- **Pulmonary:** - left 2nd space
- **Aortic**
A1 = right 2nd space.
A2 = left 3rd space. (Erb's area)

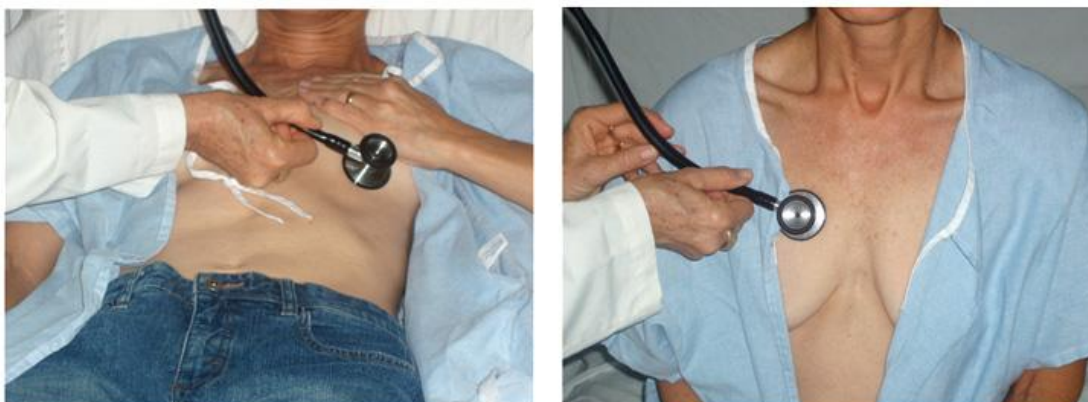
Other rare areas

- Lt Parasternal spaces:- VSD
- Left infra clavicular:- PDA.
- Posterior thorax, T2-T6: coarctation of aorta.



طبيب تقولي لوالحاله سيده
في الامتحان احط السماعه فين ؟؟؟!!!!

اقولك كده غلط والصح



c) **The manoeuvre of auscultation**

For timing examine the carotid pulse

Cone

Start with the cone of the stethoscope listens to the:

- Mitral area
- Tricuspid area

You may need special manoeuvre as:

Roll the patient on to their left lateral position and listen for the murmur of mitral stenosis.

Diaphragm

Start with mitral area

then in a Z shape direction listen to

- Tricuspid
- Pulmonary
- Then aortic areas.

You may need special manoeuvre as:

1. **Listen into the axilla area** for the murmur of mitral incompetence.
2. **Over T area** ask the patient to breath in (murmur of right side increased with inspiration)
3. **Over P area** compare between A2 and P2 (S2 of P accentuated in PH++)
4. **Over A area**, listen on the carotid a. (propagation of AS murmur) & ask the patient to set and holding breath in expiration (or hearing of AR)



In each sound comment on

- Def. & mechanism
- Causes
- Site
- Timing
- Character

Auscultatory findings

1. Heart sounds

	First heart sound	Second heart sound
Formed	a. Valvular: closure of the mitral and tricuspid valve. b. Muscular: contraction of the ventricle.	Closure of the semilunar valves (P & A) Vibration of the great vessels.
Site	Over M & T areas	Over the base (A & p)
Time	At the start of systole (carotid ascend)	At the end of systole (carotid descend).
Accentuated	• Thin person. • Tachycardia. • Children.	
	• M.S. = decreased filling of left ventricle = closure of the valve from a lower position. • Hyperdynamic states. • Short P.R. interval = decreased filling of the heart. • S. hypertension = left ventricular +++ (muscular component).	• Pulmonary hypertension. • Systemic hypertension. • Aneurysm of ascending aorta (magnification). • Hyperdynamic circulation.
Weak	Mechanical factor e.g. thick chest wall, obesity emphysema, pericardial effusion. (Distant heart sound). Shock – hypotension	
	• M.I. – T.I. • Severe myocardial diseases or heart failure. • Calcific M.S.	• A.S. – A.I. • Severe pulmonary stenosis.
Variable: atrial fibrillation		

S1: - The mitral precedes and louder than Tricuspid

S2: -

- Aortic component louder & heard all over the pericordium.
- Pulmonary component heard on pulmonary area only.

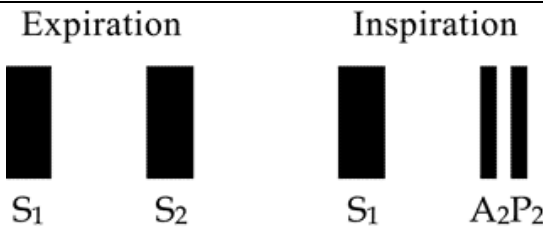
So the pulmonary component and aortic component heard over the pulmonary area and this is called physiological splitting which increase during inspiration and decrease during expiration.

Splitting of the second HS

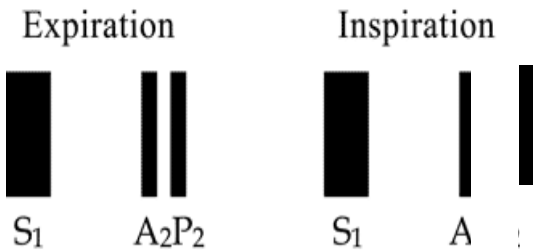
- Physiologically the 2nd HS is formed by aortic and pulmonary components
- The aortic valve is closed by pressure greater than that of pulmonary valve. So the aortic valve is closed before the pulmonary valve
- The two component are so closed so heard as a single HS

Normal (physiological) splitting:

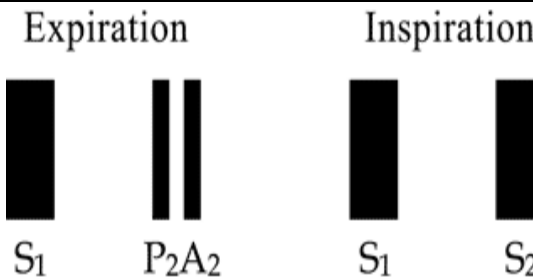
- During inspiration the pulmonary flow is increased so the closure of P valve physiologically delayed and accepted as two sounds

**Wide splitting:**

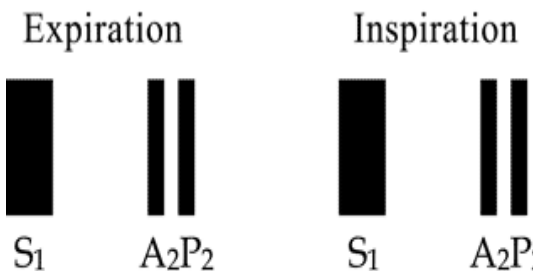
- From the start the **P valve is delayed** so accepted as two sounds
- By inspiration the pulmonary flow is increased so the splitting will be increases

**Causes:- PS / RBBB / ASD****Paradoxical splitting**

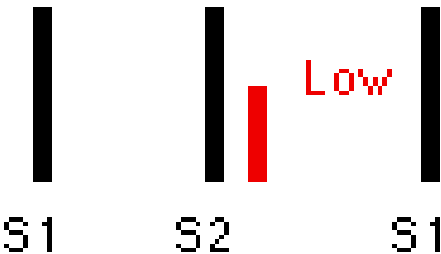
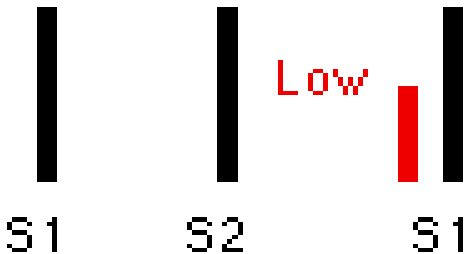
- From the start the **A valve is delayed** so accepted as two sounds
- By inspiration the pulmonary flow is increased so the splitting will disappear

**Causes:- AS / LBBB / VSD****Fixed splitting:-**

The second HS formed by two component not changed by inspiration

Causes:- A.S.D.**Single S2 :-** Sever P.S. /Sever A.S./ Single ventricle.**Closed splitting :-** PH++

2. Additional sounds

	Third heart sound	Fourth heart sound
Def	Low pitched sound heard due to gush of blood from atrium to ventricle or flabby ventricular wall	Low pitched sound heard due to forced atrial contraction against resistance
	Physiological :- in children and young adults. V. overload :- MR / AR / TR / VSD / ASD / Hyperdynamic Diminished V distinsability:- LVF \ RVF Constrictive pericarditis (Pericardial knock)	T. overload :- S. hypertension / IHD / A.S. P.S., P. embolism & PH++
Site	Mitral (left sided causes) or Tricuspid (right sided causes)	
Timing	Early diastolic (protodiastolic)	Late diastolic (presystolic)
		

Gallop: additional HS (3rd or 4th) plus tachycardia

It is so called because it is a triple rhythm resembles the sound of a galloping horse.

Causes

S3 gallop = protodiastolic gallop = LVF (M) / RVF (T)

S4 gallop = presystolic gallop = S hypertension & tachycardia

Summation Gallop: - Third & fourth sounds plus tachycardia

Causes

Hypertensive heart failure / IHD

Ejection click

Def: - Opening of the normal aortic (or pulmonary) valve is soundless while opening of the stenosed aortic (or Pulmonary) valve produce ejection click sound due to doming of this stenosed valve.

Chr.: Clicky sound.



Site	Aortic area (A.I.)	Pulmonary area
Causes	A.S. (valvular) – S. hypertension.	P.S. (valvular). – PH +++

N.B.

- No ejection systolic click with subvalvular or Calcific A.S.

Opening Snap

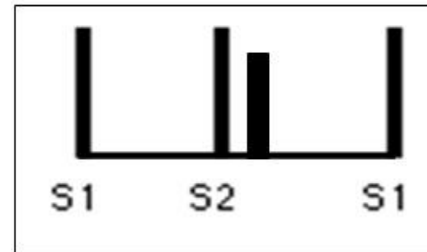
Def: - Snap sound in M.S due to rigid periphery & pliable centre of the mitral valve in M.S.

Timing: - Early diastolic / separated from S2 by the isometric relaxation phase / heard by cone

Site: - Between M & T areas.

Significance: -

- Diagnostic for M.S.
 - Non calcified.
 - Detect severity of M.S.
- (Diminished distance between O.S. & S2 = sever lesion).

**Murmur****Mechanism of Turbulence (murmur):**

- Passage of blood through
 - Stenosis (A.S. / M.S / P.S)
 - Irregularity (congenital bicuspid aortic valve)
 - Shunt (VSD / P.D.A.)
- Abnormal direction of Blood (M.R. and A.R.)
- Over blood flow (relative stenosis)
- Passage of blood into a relatively dilated structure (ejection systolic murmur in PH ++ or S hypertension)

Comment on SCRIPT

- **S**ite.
- **C**haracter.
- **R**elation to respiration & position

N.B.

- ✓ **L**eft sided heart murmurs are louder on **e**xpiration.
- ✓ **R**ight sided heart murmurs are louder on **i**nspiration.

- **I**ntensity.
- **P**ropagation.
- **T**iming.

Timing:

A.S / M.R / T.R. / VSD = systolic.

A.R./ M.S. = Diastolic.

Chr.:

A.S. = Harsh.

A.R. = Soft blowing.

M.S. = Rumbling.

M.R. = Soft (80%), harsh = (20%)

Site:

According to the diseased valve.

N.B, : A.R. murmur at A2 (Left 3rd I.C. space)

Propagation:

M.R. : Axilla / Sternum & base.

A.S. : Carotid & Apex.

Increased by:

Mitral murmur: by

1. Left lateral position.
2. Exercise.

Aortic murmur: by

1. Leaning forward.
2. Expiration.

Right. Sided murmurs: by Inspiration "**karvallo's** sign"

N.B.

Severity of the lesion detected by duration of the murmur not by grade. As duration of murmur depends on the pressure gradient across the valve

Grades (Intensity):

Intensity of a murmur is described in grades as follows:

No thrill	Grade I: Just audible in a quiet room. (Heard by an expert)
	Grade II: Quiet- (Heard by a non expert)
	Grade III: Loud without thrill. (Easily heard)
Thrill	Grade IV: Loud with thrill.
	Grade V: Very loud with the thrill. (Heard over wide area)
	Grade VI: Audible without a stethoscope. (Extremely loud)

Types of murmur

(1) Organic murmur	(2) Functional murmur
Murmur produced by structural lesions	Murmur due to functional disturbance with or without structural lesions
Most of them are diastolic	Most of them are systolic
Loud	Faint
Long	Short
Harsh (usually)	Soft
Propagated	No
With Thrill	No
With C/P	No

Pericardial Rub

Superficial, gritty, high pitched sound caused by friction of parietal & visceral layer of pericardium.

It is best heard at the left of the lower sternum with the patient breathing out using the diaphragm of the stethoscope.

Timing: To & Fro = Systolic & Diastolic.

D.D.

- Pleural rub:- disappeared by withholding breath
- Friction of stethoscope: disappeared by firm pressure
- **Crepitation**

Fine B.B.C.	Medium sized	Coarse
MS – LVF	Chest infection	Acute pulmonary oedema.

INVESTIGATIONS

Scheme for investigations

1. **Laboratory:** - Urine/ stool/ blood/ others.
2. ECG
3. Images: -
 - X ray :- Plain or with Contrast
 - Echocardiography & Doppler.
 - C.T. / MRI. (limited for pericardial diseases or tumours)
4. **Nuclear medicine as isotopic scan** (for IHD)
5. **Endoscopy**
6. **Catheterization**
7. **Biopsy**

1. Laboratory: -

Indication	Values
1. Arthralgia	For diagnosis of Rheumatic fever.
2. Fever	For diagnosis of infective endocarditis.
3. Pallor	For diagnosis of anemia.

2. ECG

Indications	Value
All cardiac cases	1. Chamber enlargement (hypertrophy). 2. Arrhythmia as A.F. 3. Ischemia (angina & M.I.).

3. Images: -

A) X ray :-

Plain		With contrast (Br. Swallow)	
Indication	Values	Indications	Values
All cardiac cases	• Chamber enlargement. • Pulmonary vasculature. • Calcific valve.	Mitral	LA ++

B) Echocardiography & Doppler

Investigation of choice: - simple, cheap, available, non invasive & highly diagnostic.

Echocardiography		Doppler
Indication	Values	Values
All cardiac cases	Aetiology: Rh(excessive fibrosis) congenital (ASD – VSD). Lesions: which valve. Severity: see later. Effect: chamber ++. Complications: - PH+++. - Calcification. - Thrombosis. Function: Ejection fraction = Stroke volume / end diastolic volume	For blood flow: Direction: Regurge. Pressure: PH +++. Velocity.

Trans-esophageal echo (TEE):

- Heart thrombus.
- Vegetation.

Assessment of severity by Echo & Doppler

MS	Valve area (normal = 4-6 cm ²) Tight MS = < 1 cm ²
AS	Pressure gradient (normal = 0 mmHg) Sever AS = > 50 mmHg
Any regurge	Degree of dilatation & EF

Catheterization

It is a long, elastic, radio-opaque, thin cord like with a central lumen.

Pathway

Right sided catheter	Left sided catheter
Any vein → I.V.C. → R.A. → tricuspid valve → Rt. Vent. → Pulmonary artery.	Any artery → aorta → aortic valve → left ventricle. Mitral valve As right sided → RA → artificial ASD → LA → M valve

Values: - As Echo & Doppler but superior in IHD

TREATMENT

Medical	Interventional	Surgical
Treatment of the cause of possible. Prophylaxis: - For rheumatic fever → to prevent recurrence of activity which is usually fatal (causes myocarditis, develops new lesion or increases severity of present lesions). - For infective endocarditis. Symptomatic: - Treatment of AF. - Treatment of HF. - Treatment of thrombosis. Curative Only in cases of AR by: Vasodilators to reduce the peripheral resistance and prevent blood regurg through the aorta.	Recent less invasive by catheterization. - In cases of stenosis → ballooning dilatation. Balloon--- valvoplast - In cases of IHD → coronary angioplasty	Indications - Failure of medical treatment. - Severe cases. - Complicated cases. Includes: - Mitral valvotomy of MS Isolated MS – non calcific valves. May be complicated by : Restenosis- iatrogenic MR. - Valve repair in regurg: usually failed. - Valve replacement: Tissue valve: - Short life span. - No anticoagulant. Used for: - Old age. - Female in child bearing period. Prosthetic valve: - Long life span. - Anticoagulant needed. May be complicated by: - Dysfunction. - Hemolytic anemia. - Endocarditis.

Heart surgery

	Opened heart surgery (OHS)	Closed heart surgery (CHS)
Technique	- Heart lung machine → used - Needs high experience.	- Not used - Simple.
Mortality	High	Low
Scar	Median sternotomy	Lateral inframammary scar
	The other cases.	Mitral valvotomy in cases of M.S. - Isolated. - Not calcified.

Mitral valvotomy:- can be done either by opened or closed heart surgery.

Valve replacement

Which valve is replaced by

- a. History.
- b. Metallic H.S.
 - S1 = Mitral.
 - S2 = Aortic.

Function of the replaced valve:

- No symptoms or signs of the disease.
- Local examination i.e. no murmur.

Complications of the replaced valve :-

- Dysfunction
- Haemolytic anemia.
- Haemolytic jaundice.
- Prosthetic valve endocarditis
- anticoagulant use

N.B.

Valve replacement should be involved in the diagnosis.

Valvular heart diseases

Mitral stenosis (MS)

Etiology

1. Rheumatic fever: (the most common cause).
2. Congenital: a rare cause.
3. Relative stenosis (not an organic stenosis)
 - Increased blood flow through the mitral valve.
 - Carey-coombs murmur in acute rheumatic valvulitis.
 - Austin flint murmur in severe aortic regurgitation.

Pathophysiology

- In mild cases:
The blood flow through the mitral valve remains normal.
No symptoms occur.
- In severe cases: (valve area less than 2 cm^2):
The blood flow through the mitral valve is decreased.
Blood stagnate in pulmonary veins (**pulmonary venous congestion**).
- Later on:
Vasoconstriction of pulmonary arterioles occurs to ↓ pulmonary congestion, but this will lead to: **pulmonary hypertension**.
- Finally:
RV enlargement & then RVF will occur secondary to pulmonary HTN.

Therefore four stages will occur in patient with MS

Stages of Mitral stenosis

- **Stage one:** "Mild or asymptomatic mitral stenosis"
The only abnormality is anatomical narrowing of the mitral valve, but the patient is fully compensated (no symptoms).
- **Stage two:** "mitral stenosis with pulmonary congestion"
The pulmonary venous pressure is elevated.
- **Stage three:** "mitral stenosis with pulmonary hypertension"
The pulmonary arterial pressure is elevated.
- **Stage four:** "mitral stenosis with right ventricular failure"

Clinical picture

There is a **latent period** of several years between the initial attack of rheumatic carditis & the development of manifestations of mitral stenosis.

Symptoms

Does the patient with MS always symptomatize???

1. **Stage one:**
No symptoms.
2. **Stage two:**
Symptoms of pulmonary congestion (but pulmonary oedema is not common).
Symptoms of low CO.
3. **Stage three:**
Symptoms of pulmonary congestions: improve.
Symptoms of Low CO: increase.
4. **Stage four:**
Symptoms of systemic congestion.

Dyspnea is the most common symptom

Signs

General:

1. **Stage one:** No signs.
2. **Stage two:** signs of **pulmonary congestion**.
3. **Stage three:** signs of **low cardiac output**.
4. **Stage four:** signs of **systemic congestion**.

Cardiac:

A. Precordial examination:

Stage one & stage two:

- ✓ Apex: normal site & slapping character.
- ✓ Diastolic thrill: ending in a palpable S1.

Stage three & stage four:

- ✓ The previous findings.
- ✓ Signs of: pulmonary hypertension.
- ✓ Signs of: right ventricular enlargement.

B. Auscultation

Stage one & stage two (over the mitral area)

1. Accentuated first heart sound: due to:

- ✚ Fibrosis of the mitral cusps.
- ✚ Forcible closure of the mitral cusps: because:
 - ❖ They are displaced downwards due to high LA pressure.
 - ❖ The mitral valve is opened as wide as possible during the diastole.

N.B. Diminished S1 in mitral stenosis denotes:

- Double mitral lesion (with predominant regurge) or
- Calcified mitral valve.

2. Mitral opening snap:

- ✓ It is **sharp & snapping** due to opening of the rigid cusps of mitral valve.
- ✓ It heard in the **early diastole**.
 - ✚ Just after S2 (separated from it by the isometric relaxation phase).
 - ✚ Just before the murmur of mitral stenosis.
- ✓ Its presence denotes **non-calcification** of the mitral valve.

3. Murmur of mitral stenosis:

- ✓ Timing:
 - ✚ Mid-diastolic pre-systolic with pre-systolic accentuation.
 - ✚ In AF, there is loss of pre-systolic accentuation (due to loss of atrial contraction).
- ✓ Character: rumbling.
- ✓ Site: at or slightly inside the apex.
- ✓ Propagation: not propagated.
- ✓ Position:
 - ✚ Best heard with the cone of the stethoscope.
 - ✚ In the left lateral position.

4. Silent mitral stenosis: (MS with no murmur) due to:

- a. High LV pressure: LVF.
- b. Low LA pressure:
 - Severe pulmonary hypertension.
 - RVF.
- c. In association with ASD.

Stage three

- The previous findings.
- Auscultatory findings of pulmonary hypertension:
 - a. Over the pulmonary area:
 - ✓ S2 is accentuated.
 - ✓ Systolic ejection click.
 - ✓ Systolic ejection murmur.
 - ✓ Soft early diastolic murmur: (Graham Steel murmur)
 - b. Over the tricuspid area:
 - ✓ S4 gallop

Stage four

- The previous findings.
- Auscultation over tricuspid area:
 - Pan systolic murmur of functional tricuspid regurge.
 - Prtodiastolic gallop (S3) due to RVF.

*Complications***1. In the mitral valve:**

- Rheumatic activity.
- Calcification.
- Infective endocarditis.

2. In the left atrium:

- a) Arrhythmias, especially AF.
- b) LA enlargement, causing symptoms:
 - On esophagus: dysphagia.
 - On left bronchus: dyspnea & cough.
 - On left recurrent laryngeal nerve: hoarseness of voice.
- c) Thrombo-embolic complications:
 - Systemic embolization: e.g. Cerebral, peripheral, renal.
 - Ball & valve embolus: leading to syncope & sudden death.

3. In the right ventricle:

- RVF.

4. In the left ventricle:

- No LVF in isolated mitral stenosis.

5. In the lung:

- Hemoptysis.
- Pulmonary infection.
- Pulmonary embolism (secondary to DVT).
- Pulmonary oedema is not common in mitral stenosis.

6. Complications of surgery.*Investigations***1. Chest X-ray:**

- a) **Stage one:** no abnormality.
- b) **Stage two:**
 - In postero-anterior view:

- Obliteration of the waist of the heart
- Double contour of the right border of the heart

} Mitralization =
LA enlargement

- Pulmonary venous congestion
 - Kerley's B lines due to interstitial oedema
- } pulmonary congestion
- In lateral view with barium:
 - Enlarged left atrium displaces the esophagus posteriorly.
- c) **Stage three & four:**
- Right ventricular enlargement.
 - Pulmonary hypertension.
 - May be calcified mitral valve.

2. ECG

- a) **Stage one:** no abnormality.
- b) **Stage two:** LA enlargement (P mitrale: broad & bifid).
- c) **Stage three & four:**
- Right atrial enlargement (P pulmonale: tall & peaked).
 - Right ventricular enlargement.

3. Echocardiography:

- The most sensitive & specific non-invasive method diagnosing MS.
- Detects the severity of stenosis.
- Detects chamber enlargement.

4. Cardiac catheterization & angiocardiography:

- Detects the severity of stenosis by measuring:
 - The valve area.
 - The pressure gradient across the valve.
- Detects chamber enlargement.

When do we say tight mitral stenosis ???

- ❖ Tight MS is diagnosed:
- **According to the stage:**
 - ✓ Stage two with dyspnea more than grade two, or
 - ✓ Stage three, or
 - ✓ Stage four.
 - **According to the echocardiography:**
 - ✓ Valve area less than 1 cm^2 .

Treatment

1. Medical:

- a) Prophylaxis against: rheumatic activity, infective endocarditis (uncommon).
- b) Symptomatic for: complications e.g. HF, AF, infection, embolization.

2. Surgical:a) Indications:

- Tight mitral stenosis (valve area is $< 1 \text{ cm}^2$).
- Marked symptoms not responding to adequate medical treatment.
- Embolization with no serious deterioration of the condition of the patient.

b) Types of operations:i. Mitral commissurotomy: closed or open:

ii. Valve replacement:

✚ By a prosthesis (tissue or synthetic).

✚ Indications:

- Calcification.
- Associated mitral regurge.
- Recurrent stenosis after commissurotomy.

c) Complications:

1. Embolization.
2. Arrhythmias.
3. Mitral incompetence.
4. Restenosis.
5. Post-cardiotomy syndrome:
 - Pleuro-pericarditis that may occur 10 – 15 days following the operation.
 - It is possibly an allergic process due to injury of the pericardium during surgery.
6. Complications of artificial valves:
 - Infective endocarditis.
 - Thrombo-embolism.
 - Mechanical dysfunction.
 - Hemolytic anemia.

3. Balloon dilatation:

- May be performed in some patients indicated for valvotomy.

Case study*Questions***A. What did you find in this patient??**

Finding suggestive of isolated mitral stenosis in sinus rhythm (or in AF).

B. What is the evidence of severity??

1. Severity of symptoms: dyspnoea (degree) paroxysmal nocturnal – hemoptysis-edema.
2. Signs of low cardiac output: fatigue – cold hands – small pulse.
3. Signs of pulmonary hypertension.
4. Duration of middiastolic murmur: the longer, the more severe.
5. Timing of the opening snap: the earlier, the more severe.

C. Explain the signs of pulmonary hypertension.

- Prominent "a" wave.
- Para sternal heave (RV hypertrophy).
- Pulsations and dullness in 2nd left space.
- Diastolic shock (palpable P2) at base.
- Loud P2 auscultation.

D. What investigations would you advise??

1. Plain X-rays of the chest to show:
 - Large left atrium: widened carina – double contour at right border.
 - Obliterated concavity on left side: dilated pulmonary artery.
 - Dilated pulmonary vessels.
 - Kerley's line.
2. ECG: bifid P wave
 - RV hypertrophy.
 - AF.
3. Echocardiography:
 - Size of mitral valve and mobility.
 - Calcification.
 - Pressures in left atrium, and other chambers.

E. What are the complications of MS???

- Atrial fibrillation.
- Systemic embolization.
- Right ventricular failure.

F. How do you manage cases with mitral stenosis???

Depends on severity:

- **Medical:** salt restriction – diuretics – digoxin, anticoagulants for AF.
Prophylaxis against infective endocarditis –
- **Surgery:** indicated in:
 - Moderate and severe cases causing symptoms.
 - Systemic embolism.
- **Surgical** procedure depends on state of valve, and pressures in left atrium, and other chambers.
 - *valvotomy: closed – balloon – or open.*
 - *Mitral valve replacement.*

Questions to score more

Q- What is the opening snap???

A- Sharp sound in early diastole produced by forcing and tensing the thickened fused mitral cusps downwards by the high atrial pressure normally, normally absent.

Q- What is its significance??

A-Cusps are still mobile – disappears if cusps are fibrosed or calcified
It reflects pressure in left atrium.
The higher, the earlier it is heard.

Q-Why is the middiastolic murmur difficult to hear in very severe mitral stenosis???

A-The blood flowing through a very narrow opening (stenosed valve) is too little to produce a significant sound (murmur).

Mitral regurgitation (MR)

Etiology

A. **Organic:**

1. Rheumatic fever: the most common cause.
2. Congenital.
3. **P**rolapse of the mitral valve (MVP)
4. **P**apillary muscle dysfunction **e.g.** CAD.
5. **I**nfective endocarditis.
6. **I**atrogenic: following mitral valvotomy.

B. **Relative:**

- Dilatation of the mitral ring secondary to LV dilatation.

pathophysiology

1. During systole:

A part of blood regurgitates from LV to LA leading to:

- Low CO.
- LA dilatation: due to increased blood volume in LA.

2. During diastole:

A large volume of blood → LV → LV enlargement which may end in LVF.

Clinical picture

Symptoms

1. No symptoms: in early cases.
2. Symptoms of low cardiac output: in late cases.
3. Symptoms of pulmonary congestion: in late cases.
4. PALPITATION.

Palpitation is the most common

Signs

General:

- No signs: in early cases.
- Signs of low cardiac output: in late cases.
- Signs of pulmonary congestion: in late cases.

Cardiac:

A. **Precordial examination:**

- Signs of LV enlargement: with hyperdynamic apex.
- Systolic thrill: over the apex.

B. Auscultation:

1. First heart sound: **weak** (muffled) due to failure of proper mitral closure.
2. Third heart sound: **present** due to excessive flow of blood from LA to LV.
3. Murmur of mitral regurge:
 - Timing: pansystolic starting with S1.
 - Character: soft or harsh.
 - Site: over the apex.
 - Propagation:
 - ✚ To the axilla.
 - ✚ To the base of the heart & medially in posterior leaflet disease.
 - Position: heard best in the left lateral position.

complications

Same complications of MS, but:

1. Infective endocarditis: is common.
2. Left ventricular failure: occurs.

*Investigations***1. Chest X-ray:**

- No abnormality: in early cases.
- Enlarged LA & LV: in late cases.
- Pulmonary congestion: in late cases.
- Calcified mitral valve especially: in double mitral lesion.

2. ECG:

- Enlarged LA (P mitrale: broad & bifid).
- Enlarged LV.

3. Echocardiography:

- Detects the severity of mitral regurgitation.
- Detects chamber enlargement.
- Detects the cause: **e.g. MVP**.

4. Cardiac catheterization & angiography:

- Detects the severity of mitral regurgitation.
- Detects chamber enlargement.
- Detects the cause: **e.g. CAD**.

*Treatment***1. Medical:**

- Same as that of mitral stenosis.

2. Surgical:

- Valve replacement.

Questions

Q-What did you find in this patient??

A-Findings suggestive of mitral regurgitation: apex shifted, and hyper dynamic – systolic thrill at apex area – pan systolic murmur maximal over apex, and propagated to axilla.

Q-What is the differential diagnosis of a pansystolic murmur???

A-1. **mitral regurgitation**: maximal over louder apex and propagated to axilla, with expiration.

2. **Tricuspid regurgitation**: maximal over lower end of sternum – with inspiration.

3. **Ventricular septal defect (VSD)**: maximal 3rd, 4th left spaces.

Q-What are the common causes of mitral regurgitation???

- Rheumatic.
- Mitral valve prolapse.
- Dilated left ventricle (dilated mitral ring): congestive heart failure – cardiomyopathy.
- Surgical complications of mitral valvotomy.

Q-What are the complications of mitral regurgitation???

- Left heart failure.
- Infective endocarditis.
- Atrial fibrillation.

Q-What is the treatment of mitral regurgitation???

- If severe: **surgery**: valve replacement usually – valve repair in minority.
- **Prophylaxis** against infective endocarditis.
- Treatment of heart failure.

Aortic stenosis (AS)

Etiology

1. Rheumatic fever.
2. Calcific.
3. Congenital:
 - ✚ it may be: valvular, subvalvular or supravalvular.
4. Hypertrophic cardiomyopathy (Idiopathic Hypertrophic Subaortic Stenosis; IHSS):
 - ✚ It produces a subvalvular obstruction in the systole due to: contraction of the hypertrophied interventricular septum.
5. Relative stenosis (not an organic stenosis):
 - a. Dilatation of the aorta:
Hypertension, atherosclerosis, aortic aneurysm.
 - b. Increased blood flow across the aortic valve:
Hyperdynamic circulation, AR.

Pathophysiology

- During systole, there is obstruction of blood flow from LV to aorta leading to:
 1. Low cardiac output.
 2. Pressure overload on LV leading to LVH & LVF.
- Normally, the aortic valve area is 3 – 4 cm². In severe AS, it is less than 0.8 cm².

Clinical picture

Symptoms

1. No symptoms: in mild cases.
2. Symptoms of low CO: in severe cases.
3. Symptoms of pulmonary congestion: due to LVF.
4. SYNCOPE: especially exertional due to low fixed CO.
5. ANGINA: due to
 - Reduced coronary blood flow: due to low CO & shortened diastole.
 - Left ventricular hypertrophy: increases the myocardial O₂ demands.
 - Associated coronary atherosclerosis: especially in calcific AS.

Signs

General:

1. Pulse:
 - Pulsus parvus et tardus (plateau pulse): *rises slowly, of small volume, returns slowly.*
 - Pulsus bisferiens: *bifid pulse occurring in double aortic lesion.*
2. BP: low SBP in severe cases.
3. Systolic thrill: over the carotid arteries.

Cardiac:**A. Precordial examination:**

1. Signs of LVH: with a heaving apex.
2. Systolic thrill: over second right space → apex & carotid arteries.

B. Auscultation:Over the aortic area:

1. **S**econd heart sound: weak.
2. **S**ystolic ejection click: due to opening of the rigid cusps.
3. **S**ystolic ejection murmur:
 - Midsystolic, harsh.
 - Maximum over second right space → apex & carotid arteries.

Over the pulmonary area:

- Reversed splitting of the second heart sound.

Over the mitral areas:

1. S4.
2. Propagated murmur of AS.

complications

1. LVF.
2. Infective endocarditis.
3. Sudden death: usually due to VF.
4. Heart block: in calcific AS due to extension of calcification to AV bundle.
5. Rheumatic activity: in rheumatic AS.

*Investigations***1. Chest X-ray:**

- No abnormality: in mild cases.
- LV: LVH.
- Lungs: pulmonary congestion when LVF occurs.
- Aorta:
 - Small aortic knuckle or post-stenotic dilatation.
 - Aortic valve calcification may be seen.

2. ECG:

- LVH.

3. Echocardiography:

- Detects the severity of stenosis by:
 - Measurement of valve area.
 - Measurement of pressure gradient across the valve.
- Detects the type of stenosis.
- Detects LVH.

4. Cardiac catheterization & angiocardiology:

- Detects the severity of stenosis by:
 - Measurement of valve area.
 - Measurement of pressure gradient across the valve.
- Detects the type of stenosis.
- Detects LVH.

	Congenital	Rheumatic	Calcific
Age	Young	Young & middle	Old
History of RF	Absent	Present	Absent
Type	Valvular or subvalvular or supravulvular	Valvular	Valvular
Associations	Congenital anomalies	Other valvular lesions	Atherosclerosis

*Treatment***1. Medical:**

- Same as that of mitral stenosis.
- Anginal attacks: may be relieved by SL nitrates.

2. Surgical: (Aortic valve replacement) indications:

- Presence of severe symptoms.
- Pressure gradient more than 50 mmHg.
- Valve area less than 0.8 cm^2 .

3. Balloon dilatation: indications:

- Children:** with congenital AS as an alternative to surgery.
- Elderly:** with severe calcific AS who are too ill to undergo surgery.

*Questions***Q-What did you find in this patient???**

- Finding suggestive of aortic stenosis.
- Heaving apex.
- Systolic thrill at the base.
- Harsh ejection systolic murmur propagated to right side of the neck.
- Small prolonged plateau pulse.

Q- How to differentiate between aortic stenosis and pulmonary stenosis???

The murmur is similar (harsh ejection systolic murmur), but differences are obvious:

- The murmur of the pulmonary stenosis is maximal over the left upper parasternal area.
- P2 is diminished or absent.
- Right ventricular hypertrophy (versus LV hypertrophy in AS).

Q-What are the causes of aortic stenosis??

- Degenerative.
- Calcific.
- Rheumatic.
- Congenital: bicuspid cusps.

Q-What are the complications of aortic stenosis???

- Left ventricular failure.
- Infective endocarditis.
- **Sudden death:** ventricular fibrillation or asystole.
- Complete heart block: extension of fibrosis and calcification to A-V bundle from neighboring aortic valve.

Q-What are the treatment of aortic stenosis???

- Balloon dilation for young patients.
- Valve replacement for elderly patients.

*Questions to score more***Q-What are the causes of ejection systolic murmur in the aortic area???**

- Aortic stenosis.
- Aortic stenosis (thickening of cusps without stenosis).
- Systolic murmur accompanying severe aortic regurgitation: "to and for" murmur.

Q-How to differentiate???

The ejection systolic murmur of aortic stenosis is distinguished by being:

- Very harsh.
- Propagated to the carotids.
- Accompanied by systolic thrill.

Aortic regurgitation (AR)

Etiology

1. Rheumatic fever: **the most common cause**.
2. Infective endocarditis.
3. Congenital.
4. Dilatation of the ascending aorta as in :
 - a. Syphilitic aortitis: syphilis produces dilatation of the aortic ring.
 - b. Severe hypertension.
 - c. Ankylosing spondylitis.
 - d. Aortic aneurysm: with dissection.
 - e. Marfan syndrome.

Pathophysiology

Regurgitation of blood from the aorta to the LV in diastole leads to:

1. Increased LV stroke volume, this results in:
 - a. **Increased SBP.**
 - b. Peripheral VD which (together with regurgitation) will **decrease the DBP.**
 - c. **↑ SBP & ↓ DBP → wide pulse pressure** causing peripheral signs of AR.
2. Volume overload on the LV leading to LV dilatation & later on LVF.

Clinical picture

Symptoms

1. Generalized **body throbbing**: due to increased arterial pulsation.
2. **Palpitation**: due to forcible LV contraction.
3. Symptoms of pulmonary congestion: when LVF occurs.
4. Angina pectoris: "two types of angina occur in aortic regurge"
 - **Classic angina of effort** :
Decreased DBP: reduces coronary filling.
 - **Angina of Lewis**:
Nocturnal & associated with autonomic disturbance **e.g.** sweating & tachycardia.

Signs

General: "Peripheral signs of aortic regurge"

4 head and neck, 3 upper limb and 3 lower limb

- 4 head and neck
 1. De-Musset sign: nodding of the head.
 2. Corrigan's sign: prominent carotid pulsations.
 3. Systolic thrill: over the carotid arteries.
 4. Arteriolar pulsation: seen by fundus examination

- **3 upper limb**

1. Water hammer pulse: Raises rapidly, of big volume, collapses rapidly.
2. Blood pressure
 - a. Wide pulse pressure: exaggerated differences between systolic and diastolic blood pressure.
3. Capillary pulsations: detected in nail bed, lips or ear lobule.

- **3 lower limb**

1. Pistol shots: loud booming sounds heard with each pulse beat over the arteries (especially femoral) due to sudden distension of collapsed arteries.
2. Duroziez's sign:
 - It consists of systolic & diastolic murmurs over the femoral artery if it is slightly compressed with the stethoscope bell.
 - The systolic murmur is due to the rapid flow of blood to periphery, while the diastolic murmur is due to rapid regurge of blood to the heart.
3. Hill's sign:
 - Exaggerated difference between SBP in LLs & ULs: more than 50 mmHg.
 - Normally, SBP in LLs is higher than in ULs: by about 10 – 20 mmHg.

Cardiac

A. Precordial examination:

- Signs of LV enlargement: with hyperdynamic apex.
- No thrill over the aortic area: in isolated AR.

B. Auscultation:

- **Over the aortic area:**

- a. Normal second heart sound.
- b. Murmur of AR:
 - Timing: early diastolic.
 - Character: soft blowing, decrescendo.
 - Site: maximum over the third space.
 - Propagation: to the apex.
 - Position: **best heard with the diaphragm of the stethoscope, the patient is:**
 - Sitting up.
 - Leaning forward.
 - Holding his breath in forced expiration.
- c. Soft ejection systolic murmur:
 - Due to ↑ blood flow across the aortic valve (relative AS).
- **Over the mitral area:**
 - a. S3.
 - b. Propagated murmur: of AR.
 - c. Pan systolic murmur: of functional MR.

d. Austin Flint murmur: (mid-diastolic) due to:

- Elevation of anterior leaflet of the mitral valve by the regurgitant blood from the aorta causes relative MS.

Complications

1. Rheumatic activity.
2. Infective endocarditis.
3. LVF.

Investigations

1. Chest X-ray: "Aortic configuration (**Boot-shaped heart**)"

- LV enlargement & dilated aorta.

2. ECG:

- LV enlargement.

3. Echocardiography:

- Detects the severity of the lesion.
- Detects chamber enlargement.

4. Cardiac catheterization & angiocardiology:

- Detects the severity of the lesion.
- Detects chamber enlargement.

Treatment

1. Medical:

- Same as that of mitral stenosis.
- Syphilis: anti-syphilitic treatment.

2. Surgical: (aortic valve replacement) indications:

- a. Presence of severe symptoms.
- b. Progressive cardiomegaly.
- c. Declining LV functions.

Questions

Q-What did you find in this patient???

Findings suggestive of aortic regurgitation:

- Shifted hyperdynamic apex.
- Early blowing diastolic murmur.
- Corrigan's sign – waterhammer pulse.
- Very low diastolic pressure.

Q-What is the cause of pallor in this patient???

Vasoconstriction: compensatory in severe aortic valve disease.

The patient is not anemic: color of tongue is normal.

Q-How can you differentiate aortic regurgitation (AR) from pulmonary regurgitation (PR)??

The early diastolic murmur is identical in both conditions.

Distinction is made by noting:

- Peripheral arterial phenomena: confirm AR.
- Manifestations of pulmonary hypertension: confirm PR.

Q-What are the causes of aortic regurgitation???

- Rheumatic.
- Marfan's syndrome.
- Infective endocarditis.
- Syphilitic aortitis.

Q-What is Marfan's syndrome???

Genetic disorder:

- Tall thin built.
- Long extremities.
- Arachnodactyly (long thin fingers with pads in between).
- High arched palate.

Q-how do you assess severity of aortic regurgitation??

1. Apex beat: noting the extent of displacement and degree of hyperkinetic pulsation.
2. Peripheral phenomena.
3. B.P.: how wide is the pulse pressure.

Q-What are the complication of AR???

- Heart failure.
- Infective endocarditis.

Q-What is the treatment??

- Prophylactic antibiotics.
- Valve replacement: severe cases.

Tricuspid regurgitation (TR)

Etiology

1. Functional: **the most common cause** due to dilatation of the tricuspid ring secondary to RV dilatation.
2. Organic: **rare:**
 - Rheumatic fever.
 - Infective endocarditis.
 - Congenital.
 - Carcinoid syndrome.

Pathophysiology

During systole, blood regurgitates from RV to RA causing:

- Low CO.
- RA enlargement.
- RV enlargement.
- RVF (systemic congestion).

Clinical picture

Symptoms

1. Systemic congestion.
2. Low CO.

Signs

General:

1. Systemic congestion including:
 - *Congested pulsating neck veins: with systolic expansion.*
 - *Enlarged tender pulsating liver.*
 - *Ascites **before** oedema of LLs (Ascites precox).*
 - *Mild jaundice & peripheral cyanosis (cyano-icteric face).*
2. Low CO.

Cardiac:

- A. Precordial examination:
 - RA & RV enlargement.
 - Rarely: systolic thrill over tricuspid area.
- B. Auscultation: "over tricuspid area"
 - a. Weak muffled S1.
 - b. S3.

c. Pan systolic murmur.

- Timing: pan systolic.
- Character: soft or harsh.
- Site: tricuspid area.
- Propagation: to the apex, but not to the axilla.
- Caravallo's sign: murmur increases with inspiration being of right side origin.

Investigations

1. **Chest X-ray:**

- RA & RV enlargement.

2. **ECG:**

- RA & RV enlargement.

3. **Echocardiography:**

- Diagnostic.

4. **Cardiac catheterization & angiocardiology:**

- Diagnostic.

Treatment

1. **Medical:**

- Treatment of right sided heart failure.

2. **Surgical:**

- Valve replacement.

Questions

Q-What did you find in this patient???

Findings suggestive of tricuspid regurgitation:

- Increased JVP with marked systolic expansion (V wave).
- Parasternal hyperdynamic pulsations (marked dilatation of right ventricle).
- Pansystolic murmur at lower end of sternum, increased with inspiration.
- Enlarged pulsating liver.

Q- What is the etiology of tricuspid regurgitation???

Most cases are functional: dilated ring due to congestive heart failure.

The heart with multivalvular lesions what to do??

First

Valuable information may be observed in JVP and in arterial pulse:

- Raised JVP with prominent systolic wave = tricuspid regurgitation.
- Corrigan's sign – waterhammer = aortic regurgitation.
- Pulsus bisferiens (double strokes): aortic stenosis + aortic regurgitation.

Second

Pick the very significant abnormal signs suggestive of one lesion and then confirm by auscultation *i.e. one lesion by one.*

1. **Hyperdynamic apex:** either aortic or mitral regurgitation:
 - **Aortic regurgitation:** after noting Corrigan's signs (vigorous pulsation of the carotids) – waterhammer pulse: big pulse pressure with very low diastolic < 60 mm. Then confirm by listening carefully to soft blowing early diastolic murmur at the lower right parasternal area.
 - **Mitral regurgitation:** check by feeling a **systolic thrill** at the apex (patient lying on his left side after exercise)
Confirm by listening to pansystolic murmur maximal over the mitral.
A mid diastolic murmur may also be heard (mitral stenosis).
2. **Signs of pulmonary hypertension:**
 - Left parasternal heave. (RV +++).
 - Pulsations and dullness in second left space.
 - Diastolic shock. (accentuated P2).
 - Prominent "a" wave.

Practically mitral stenosis is highly suspected.

- Confirm then by listening carefully to a mid diastolic murmur commonly with a presystolic accentuation.
 - Otherwise suspect other causes of pulmonary hypertension.
3. **Heaving apex:** aortic stenosis or severe hypertension.
Aortic stenosis check with feeling a systolic thrill: patient sitting and leaning forwards, holding his breath in expiration.
Confirm by listening to a harsh ejection murmur over aortic area propagated to the root of neck.
Then feel the arterial pulse and revise its character: slow and prolonged.
 4. **Tricuspid regurgitation:** most easily suspected by noting:
 - The distended neck veins with marked systolic expansion (v wave).
 - Expansile liver (bimanual palpation).
 - *Confirm* by listening over tricuspid area for a pansystolic murmur which is increased by inspiration.

Valve lesion	Most important cause	Most important symptom	Most important sign	Chest X-ray	ECG
MS	Rheumatic	Pulmonary congestion esp. <u>dyspnea</u>	• Accentuated S1. • Mid-diastolic rumbling <u>M</u> over apex. • Pulmonary hypertension.	• Mitralization. • Pulmonary congestion	• P-mitrale. • P-pulmonale. • AF.
MR	Rheumatic	<u>Palpitation</u>	• Pan systolic <u>M</u> & thrill over the apex propagated to axilla	• LAE. • LVE.	• LAE. • LVE.
AS	Rheumatic Calcified Congenital	Low CO esp. <u>Angina & syncope</u>	• Heaving apex. • Harsh ejection systolic <u>M</u> & thrill "over the second right space → carotid"	• LVE. • Small aortic knuckle or • Post-stenotic dilatation.	• LVE.
AR	Rheumatic Syphilitic	Throbbing palpitation	• Peripheral signs. • Hyper dynamic apex. • Early diastolic soft blowing <u>M</u> "over the third left space"	• Boot shaped heart.	• LVE.
TS	Rheumatic	Systemic congestion	• Giant a-wave. • Mid-diastolic rumbling <u>M</u> over tricuspid "↑ with inspiration"	• RAE.	• RAE.
TR	Functional	Systemic congestion	• Systolic expansion in neck vein. • Pansystolic <u>M</u> over tricuspid "↑ with inspiration"	• RAE. • RVE.	• RAE. • RVE.

Cardiology (long case)

A. History

Personal history

Mr. xxxx 41 years old, from Giza, driver, married 21 years ago and has 3 daughters, the youngest is 2 years old, he is moderate smoker.

- **He is complaining of:** shortening of breathing of 1 week duration.
- **History of present illness:** patient was quiet well until he suffered from an exertional dyspnea of gradual onset and progressive course with 10 years duration. This dyspnea increased by exercise and decreased by rest, treated by digoxin but recurrent, No orthopnea or P.N.D.

No cough, haemoptysis or recurrent chest infection.

- 3 years later he developed regular palpitation of gradual onset and progressive course, increased by exercise & decreased by rest.
- 2 months ago palpitation became irregular without precipitating or relieving factors.
- No symptoms of systemic venous congestion.
- No cyanosis, no symptoms of low cardiac output or syncope.
- No chest pain, pressure manifestations, B.P. changes, fever or embolic manifestations.
- No symptoms of other systems affection.
- Patient investigated by E.C.G., chest X-ray, Echo cardiography and Doppler and treated by digoxin, marivan and lasilacton.
- **Past history** of rheumatic fever at age of 7, manifested by fever, polyarthritis and skin rash, investigated by blood analysis and treated by aspirin long acting penicillin every month without compliance.
 - No history of surgical operation, allergy, or drugs intake.
- Irrelevant family history.

B. General examination

1. Patient with an average general condition.
2. Mentality: patient is fully conscious, oriented by time, place and persons, with good mood and memory; he is co-operative with an average inelegancy.
3. Average built.
4. Patient lies comfortable in bed.
5. Face:
 - Body temperature = 37.1 °C.
 - No pallor, jaundice or cyanosis.

6. Neck:

- Not congested neck veins.
- Normal carotid pulsation.
- Central trachea.
- No thyroid or lymph node enlargement.

7. Upper limbs:

- Pulse: 85 beats / min., irregular, variable volume, equal in both sides, pulse deficit > 10 / min., condition of blood vessels are normal with palpable dorsalis pedis.
- Blood pressure = 110 / 70.
- No hand clubbing.

8. Lower limbs:

- No lower limb edema.
- Intact peripheral pulsations.

c. Local examination**Inspection:**

- No precordial bulge.
- No scars.
- No dilated vein or pigmentations.
- Visible apical pulsation.

Palpation:

- Apex
 - ✓ Site: left 5th I.C. / M.C.L.
 - ✓ Area: localized.
 - ✓ Character: hyper dynamic.
 - ✓ Thrill: systolic thrill.

Percussion:

- Hepatic dullness in 5th I.C. space.
- No dullness outside right border of the heart.
- No dullness on base of the heart.
- No dullness outside the apex.
- No sternal dullness.

Auscultation:

- Apex
 - ✓ Variable S₁.
 - ✓ Murmur:
 - Pansystolic.
 - Soft.
 - On apex.
 - Propagated to axilla.
 - ↑↑ by exercise.

- Tricuspid
 - ✓ Variable S_1 .
 - ✓ No murmur.
- Base
 - ✓ Normal S_2 .
 - ✓ No murmur.

No additional sounds or crepitation.

Diagnosis

A case of rheumatic valvular heart disease, most probably M.S. & M.R.
Patient is compensated but complicated by A.F.